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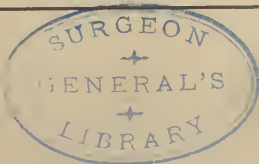
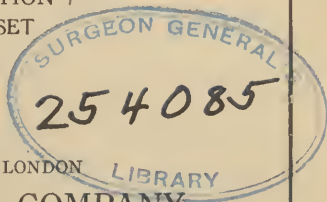
Professor of Applied Therapeutics in the University of Pennsylvania;
Visiting Physician to the Philadelphia General Hospital

“— is an arch wherethro’
Gleams that untravell’d world whose margin fades
Forever and forever when I move.”

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PHILADELPHIA AND LONDON
W. B. SAUNDERS COMPANY

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PREFACE TO THE ELEVENTH EDITION

The text of the eleventh edition of this manual has been carefully revised throughout and much new material has been added to it to supplement or replace the old. Many sections have been entirely rewritten and articles dealing with chronic family jaundice, arteriosclerotic kidney, secondary hyperthyroidism, thromboangiitis obliterans, tumors of the lungs and pleura, botulism, epidemic encephalitis, progressive lenticular degeneration, and subacute combined sclerosis of the spinal cord have been added. In making these changes the original object with which the book was written has been steadily borne in mind and no departure from its elementary character has been attempted.

A. A. STEVENS.

September, 1923.

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DISEASES OF THE DIGESTIVE SYSTEM

GENERAL SYMPTOMATOLOGY

THE TEETH AND GUMS

Delayed dentition and the eruption of badly formed teeth may result from rickets, congenital syphilis, or cretinism.

Hutchinson's Teeth.—The lateral incisors of the upper jaw are pegged, and the central incisors of the same jaw have convex sides and crescentic notches on their cutting-edges. These peculiarities indicate hereditary syphilis, and are noted only in the permanent teeth.

A *blue line* on the gums near the insertion of the teeth usually indicates chronic lead-poisoning. Copper- and silver-poisoning occasionally produce similar lines.

Spongy, bleeding gums are often associated with scurvy. Swelling of the gums, with tenderness and salivation, occurs in mercurial poisoning.

THE TONGUE

Fur on the Tongue.—This consists for the most part of accumulated epithelial cells, particles of food, and micro-organisms, and occurs in a great variety of diseases, both local and general.

A *light, uniform coat* is often noted in health, particularly in those who sleep with the mouth open. Other casual conditions are: (1) Febrile diseases. (2) Dyspepsia. (3) Catarrhal conditions of the nose and throat. Very little

diagnostic significance is to be attached to the appearance of the tongue in diseases of the stomach.

Circumscribed furring often indicates local disturbance, as a jagged tooth or tonsillitis.

Unilateral furring may result from disturbed innervation, as in conditions affecting the second and third branches of the fifth nerve. It has been noted in neuralgia of those branches, and in fractures of the skull involving the foramen rotundum.

The dry, brown and fissured tongue is noted in asthenic fevers, as typhoid fever, pneumonia, and septicopyemia.

A red, beefy tongue is sometimes noted in chronic wasting diseases. It is of quite frequent occurrence in dysentery and in diabetes.

The "strawberry tongue" is characterized by a white fur, through which project bright red and prominent papillæ. It is seen in the early stage of scarlet fever.

DISCOLORATION OF THE TONGUE

Black Tongue (Nigrities Linguae).—This is a chronic affection of the tongue, characterized by the appearance of black patches on the center of the dorsum, with great prolongation of the filiform papillæ. The etiology is obscure.

Bluish-black discoloration of the tongue is observed in Addison's disease.

Leukoplakia Buccalis.—In this condition there are slightly elevated, smooth, opaque, whitish plaques on the lingual or buccal mucous membrane. There are no subjective symptoms. Excessive smoking is a common cause. Syphilis appears to be a factor in some cases. It is sometimes associated with chronic affections of the skin, notably psoriasis. Epithelioma of the mouth is not an uncommon sequel.

ENLARGEMENT OF THE TONGUE

Macroglossia, or chronic enlargement of the tongue, occurs in myxedema (cretinism), acromegaly, and occasionally in syphilis.

TREMOR OF THE TONGUE

Trembling of the tongue is noted in many conditions; it is particularly marked in asthenic fevers (typhoid), in alcoholism, and in paretic dementia.

FISSURES ON THE TONGUE

Fissures on the tongue may result from severe glossitis, syphilis, carcinoma, tuberculosis, or the impact of a jagged tooth.

SCARS ON THE TONGUE

Scars on the tongue often result from syphilitic lesions or from the tooth wounds of epilepsy.

FETOR OF THE BREATH

This is often due to local inflammation, as chronic rhinitis, pyorrhea alveolaris, tonsillitis, etc., to the retention of decomposing food, to caries of the teeth, to certain lung diseases, especially gangrene and bronchiectasis, to dyspepsia, and to the ingestion of certain foods or drugs.

THE APPETITE

Bulimia, or *inordinate appetite*, is a common symptom in nervous dyspepsia, hysteria, diabetes, and in certain insanities, notably in paretic dementia. It may be due to intestinal parasites.

Anorexia, or *loss of appetite*, is a symptom common to many conditions.

Parorexia is a craving for special articles of diet. *Pica* is a desire for articles that are not foods. These perversions of the appetite are noted particularly in hysteria, insanity, chlorosis, pregnancy, and helminthiasis.

DYSPHAGIA

Dysphagia, or difficult swallowing, may result from: (1) Local inflammation, especially tuberculous ulceration of the

throat or larynx. (2) Stricture resulting from the cicatrization of an ulcer (corrosive poisons, syphilis, typhoid fever). (3) Cancer of the esophagus. (4) Spasm of the esophagus (simple cardiospasm, hydrophobia, hysteria, etc.). (5) A foreign body. (6) Pressure on the esophagus (aneurysm, mediastinal tumor, enlarged glands, pericardial effusion). (7) Diverticula of the esophagus. (8) Paralysis, local, as in diphtheritic neuritis, or central, as in bulbar disease.

VOMITING, OR EMESIS

Etiology.—(1) Toxic, from certain drugs, uremia, acidosis, general infections, etc. (2) Organic diseases of the stomach, such as ulcer, carcinoma, gastritis, and congenital stenosis of the pylorus. (3) Intracranial disease, such as tumor, meningitis, etc. (4) Excitation of the semi-circular canals, as in sea-sickness, swinging, etc. (5) Reflex, as from uterine displacements, appendicitis, nephroptosis, biliary colic, etc. (6) Intestinal obstruction. (7) Persistent coughing, as in pertussis, pulmonary tuberculosis, pulmonary congestion from cardiac disease. (8) Certain neuroses such as hysteria and migraine. The vomiting of pregnancy may be reflex or toxic. Periodic vomiting may occur as a feature of the gastric crises of tabes dorsalis, as an expression of a transitory antointoxication (recurrent vomiting of infants), or as a neurosis.

THE VOMIT

Watery or mucous vomit is noted in chronic gastritis, in certain forms of nervous dyspepsia, in cerebral disease, and after persistent emesis, as in cholera.

Bilious or green vomit is not diagnostic of any special condition; it may occur in any case in which there are persistent vomiting and retching.

Bloody Vomit (Hematemesis).—For causes, see page 66. If present in large amount, the blood can usually be recognized by the unaided eye; small amounts may be detected by the microscope, by the spectroscope, or by chemical tests.

Purulent vomit may result from the rupture of an abscess into the esophagus or stomach or from phlegmonous gastritis.

Fecal vomit (*stercoraceous vomit*) indicates intestinal obstruction or a gastro-colic fistula, the result of ulcer or cancer. It is recognized by its odor and appearance.

Profuse Vomit.—The ejection of enormous quantities of frothy fermented material at intervals is highly significant of gastric dilatation.

Vomiting without nausea, distress, or other gastric phenomena occurs in certain neuroses of the stomach, in hysteria, uremia, and in brain disease, as tumor or as a precursor of apoplexy.

EXAMINATION OF THE GASTRIC CONTENTS

The *test-breakfast* of Ewald and Boas consists of a roll and from 10 to 14 fluidounces of water or weak tea. It is given in the morning on an empty stomach, and is removed in one hour by aspiration or expression. The roll should be thoroughly masticated. This breakfast affords a satisfactory means of determining the secretory activity of the stomach. Riegel's *test-meal*, however, is better adapted to determining the total functional activity of the stomach. It consists of a plate of meat-broth, a beef-steak weighing from 5 to 7 ounces, $1\frac{1}{2}$ ounces of mashed potatoes, and a roll. The contents are removed in three or four hours after the ingestion of the meal.

Test for Free Acids.—Filter-paper soaked in a solution of Congo-red and dried turns blue in the presence of free acids. A saturated alcoholic solution of tropeolin ∞ turns from a brownish yellow to a dark brown when brought in contact with fluids containing free acids.

Qualitative Tests for HCl.—Günzburg's phloroglucin-vanillin test will react with 1 part of HCl in 15,000 parts of water. The solution consists of 2 parts of phloroglucin, 1 part of vanillin, and 30 parts of absolute alcohol. When a few drops of this solution are heated with an equal quantity of the filtrate contained in a porcelain dish, a beautiful red

color appears at the margin of the fluid. Boas states that the test is still more delicate when 100 parts of 80 per cent. alcohol are substituted for the 30 parts of absolute alcohol.

Boas' resorcin-sugar test gives a similar reaction. The reagent consists of 5 parts of resorcin, 3 parts of sugar, and 100 parts of diluted alcohol.

Total Acidity.—This is determined by allowing a decinormal alkali solution (water, 10 c.c.; potassium hydrate, 56 mg.) to flow from a buret, drop by drop, into a beaker containing 10 c.c. of filtered juice, to which have been added as an indicator two drops of a 1 per cent. alcoholic solution of phenolphthalein. The test is completed when the red color produced no longer disappears on shaking the solution. Ten c.c. of normal gastric juice usually require from 4 to 6.5 c.c. of the standard alkali solution.

Since 1 c.c. of the alkali solution is equivalent of 0.00364 gram of HCl, it follows that the percentage of the latter in a given specimen will equal the number of cubic centimeters of the alkali solution required multiplied by 10, and again by 0.00364

Quantitative Test for Free Hydrochloric Acid.—*Mintz's Color Method.*—To 10 c.c. of the filtrate add a decinormal solution of sodium hydrate from a buret until a droplet (removed with a platinum loop) of the fluid no longer reacts with Günzburg's reagent. The number of cubic centimeters of the alkaline solution used, multiplied by 10 and then by 0.00364, gives the percentage of free hydrochloric acid. This method, which is sufficiently accurate for clinical purposes, is based upon the supposition that the alkali first unites with the free acid before it affects the acid in organic combinations.

Test for Lactic Acid.—The presence of lactic acid in the stomach-contents simply indicates the existence of subacidity and of stagnation. These two conditions are never so constantly present nor so intense as in carcinoma (Riegel). When free HCl is present in sufficient quantities, it is unnecessary to test for lactic acid.

Uffelmann recommends a mixture of 10 c.c. of a 4 per cent. carbolic acid solution and 20 c.c. of distilled water, to which is added one drop of the official liquor ferri chloridi. This makes a clear amethyst-blue solution. The reagent must always be prepared at the time of making the test. If the solution turns yellowish green on the addition of filtered gastric contents, the presence of lactic acid is demonstrated. As the blue serves only as a contrast color, a very dilute solution of iron chlorid alone (one drop of liquor ferri chloridi in 50 c.c. of distilled water) suffices. As other substances, such as sugar, alcohol, acid phosphates, etc., give a somewhat similar reaction, the test is made more reliable by exhausting the gastric filtrate with pure ether (10 vol.), evaporating the ether, and adding the reagent to an aqueous solution of the residue.

Test for Pepsin.—If free hydrochloric acid is present, the presence of pepsin in sufficient quantities may be assumed. To determine the presence of pepsin, pour 10 c.c. of filtrate into a test-tube. If free HCl is absent, add a sufficient quantity of acid to cause the appearance of the Congo reaction. Drop discs (1.5 mm. thick and 10 mm. in diameter) of hard-boiled egg into the mixture, and put the test-tube into the thermostat at 100° F. If sufficient pepsin is present, the discs will be completely dissolved in from one-half to one hour.

Test for Carbohydrates.—When starch digestion is arrested too early, as in cases of excessive secretion of HCl, Lugol's solution gives a blue or purple coloration with the gastric contents. Complete absence of color reaction indicates very active starch digestion (subacidity).

The Absorptive Power of the Stomach.—This is usually determined by the time required for free iodine to appear in the saliva after the ingestion of potassium iodid. The saliva is received on filter-paper impregnated with starch; a drop or two of fuming nitric acid is then added, and the appearance of a blue color proclaims the presence of iodine. Normally the saliva should yield the reaction for iodine in from ten to fifteen minutes after the ingestion of a capsule containing 0.1 gram of

potassium iodid. Care must be taken that none of the drug adheres to the outside of the capsule. This test cannot be regarded as being very reliable.

The Motor Power of the Stomach.—Ewald has suggested the use of salol, which escapes from the stomach into the intestine, where it is broken up into salicylic acid and phenol. Normally salicyluric acid appears in the urine from forty to seventy-five minutes after the ingestion of 1 gram of salol. Filter-paper moistened with urine containing salicyluric acid assumes a violet color when treated with a 10 per cent. ferric chlorid solution.

Riegel's test is more reliable. Normally, no food should be removed from the stomach by lavage 7 hours after a test-meal of broth, beef-steak, mashed potatoes, and a roll (see p. 21). Large residues after 12 hours almost always indicate mechanical obstruction at or near the pylorus. The x-ray is also a valuable means of determining the emptying time of the stomach. After a meal of gruel (300–500 grams) and barium sulphate (100–120 grams) the stomach is normally empty, as a rule, in from 3 to 5 hours.

ACIDITY OF THE GASTRIC CONTENTS

Normal acidity is due to hydrochloric acid, but other acids are frequently formed during the digestive process, such as lactic, butyric, and acetic acids. The quantity of hydrochloric acid in normal gastric juice varies from 0.14 to 0.2 per cent., more acid being secreted after a heavy meal than after a light one. The amount of acidity per 100 c.c. of gastric contents is usually indicated by the number of cubic centimeters of $\frac{1}{10}$ normal sodium hydrate solution that are required to bind the acid. The average figures for *total acidity* are 50 to 75, and for *free* hydrochloric acid, 20 to 40.

Hyperacidity (hyperchlorhydria) results from a variety of causes. Mental overexertion, and the persistent use of highly seasoned foods are general predisposing factors. It is frequently present in migraine, in neurasthenia and in hysteria,

It may attend the gastric crises of locomotor ataxia. It may result from the abuse of tobacco. It is present, as a rule, in ulcer of the stomach. It is a common symptom in chlorosis. It sometimes occurs in cholelithiasis and in nephrolithiasis.

Subacidity and Anacidity (Hypochylia Gastrica and Achylia Gastrica).—Decreased secretion of gastric juice often occurs in gastric carcinoma, chronic gastritis, atrophy of the gastric tubules, passive congestion of the stomach, pernicious anemia, gout and hyperthyroidism.

RUMINATION, OR MERYCISMUS

Rumination is a condition, rarely observed in man, in which the food is regurgitated from the stomach and subjected to a second mastication. It is the result of a neurosis, and is generally found in association with hysteria, epilepsy, neurasthenia, or idiocy. It is sometimes hereditary or acquired by imitation.

HICCUP

Hiccup, or singultus, results from a clonic spasm of the diaphragm, and is often noted as a temporary condition after too rapid eating or drinking. Persistent hiccup is sometimes present in extreme exhaustion following acute or chronic diseases. It may also result from irritation of the phrenic nerve, as from the pressure of a thoracic aneurysm. It may be reflex from stomachic, hepatic, intestinal, or peritoneal disease. It may be due to hysteria. It is occasionally epidemic and apparently in relation with influenza.

THE STOOLS

Blood in the Stools (Enterorrhagia or Melena).—The blood is nearly normal in appearance after profuse hemorrhages, or when it has been quickly discharged, as in piles and fissure. Retained blood imparts a black or tarry appearance to the stools.

Melena may result from: (1) Traumatism; (2) acute inflammation of the bowels, as in enteritis and dysentery; (3) passive congestion, as in chronic heart and liver disease; (4) rectal schistosomiasis (flake infection); (5) blood dyscrasia, as in scurvy, purpura, yellow fever, etc.; (6) rupture of an aneurysm; (7) ulcers in the intestines, as peptic, typhoid, dysenteric, tuberculous or malignant ulcers; (8) intussusception; (9) the passage of blood from the stomach in hematemesis; (10) hemorrhagic infarction of the bowel from embolism or thrombosis of the mesenteric vessels; (11) piles, fissure, fistula.

Occult Bleeding.—This term is applied to hemorrhages of such small proportions that the blood can be detected only by chemical tests, the microscope, or the spectroscope. When all other sources of blood can be excluded (among others the ingestion of raw meat), occult bleeding is an important indication of ulcer or cancer of the digestive tract.

Watery or serous stools are noted in choleraic diseases, in nervous diarrhea, in the colliquative diarrhea which terminates wasting diseases, in severe enteritis, and in corrosive poisoning, as by arsenic or antimony.

Green stools may be due to the consumption of much green vegetable matter (chlorophyl); to the use of calomel or other drugs which prevent the transformation of bile-pigment into urobilin; or to diarrhea, owing to the presence of bacterial pigments or undecomposed bile-pigment.

Black stools follow intestinal hemorrhage and the use of certain drugs, as charcoal, bismuth, iron, tannin, etc.

Red stools usually indicate blood, but they may occur also after the administration of hematoxylin (logwood).

Clay-colored or grayish-white stools accompany obstructive jaundice. They are seen without icterus in a great variety of intestinal disorders, probably as a result of the faulty reduction of the bile-pigment. Stools may also be light colored from the presence of a large amount of fat.

Mucous stools are noted in intestinal catarrh, particularly when the lower bowel is affected, as in ileocolitis and colitis. They also occur with mucous colic.

Fatty stools result from the ingestion of large quantities of fats, from the absence of bile, from various diseases of the upper bowel, and from chronic pancreatic diseases.

Purulent stools result from fistula in ano, dysenteric, syphilitic, or malignant ulceration, or the rupture of abscesses into the bowel, as prostatic and pelvic abscesses.

Lienteric stools, those which contain much undigested food, are noted in inflammatory conditions of the stomach and upper bowel.

ABDOMINAL DISTENTION

Causes.—(1) Enlargement of the various organs from tumors or other causes. Recognized by the history, irregular enlargement, and special symptoms referable to the organ affected. (2) Ascites. Recognized by movable dulness with superincumbent tympany, and fluctuation. (3) Chronic peritonitis (tuberculous or cancerous) with effusion. Recognized by the history, progressive emaciation, presence of a primary lesion elsewhere, and detection of tumor-like masses, with, perhaps, pain and tenderness. (4) Tympanites. Recognized by universal tympany on percussion. (5) Pregnancy. Recognized by suppression of menses, morning emesis, pigmentation of mammary areola, softening of the cervix, intermittent uterine contractions, etc. (6) Distention of the bladder. Recognized by the history, location of dulness, and results of catheterization.

DISEASES OF THE MOUTH, TONSILS, PHARYNX AND ESOPHAGUS

STOMATITIS

Definition.—Inflammation of the mouth.

Varieties.—The following are the most important: (1) Acute catarrhal stomatitis. (2) Aphthous stomatitis. (3) Ulcerative stomatitis. (4) Mycotic stomatitis (thrush). (5) Gangrenous stomatitis. (6) Mercurial stomatitis. (7) Gonorrheal stomatitis.

Acute Catarrhal Stomatitis.—This form is often caused by mechanical, thermic, or chemical irritation. It is a frequent accompaniment of specific fevers and wasting diseases. In children it is commonly seen in association with digestive disturbances, the result of improper food or faulty hygiene, especially of the mouth.

The chief *symptoms* are restlessness, disinclination to nurse, salivation, slight elevation of temperature, and fetor of breath, with reddening and swelling of the buccal mucosa.

Treatment.—The cause must be removed. In infants, cleansing of the mouth and of the mother's nipples, or of artificial nipples, if these be used, is imperative. Digestive disturbance should receive careful attention. Cool antiseptic mouth-washes, as a 3 per cent. solution of borax or boric acid with glycerin, are serviceable. In obstinate cases the mouth may be lightly painted with a weak solution of silver nitrate (3 or 4 grains to the ounce).

Aphthous Stomatitis.—This form is usually seen in children. Digestive disturbance is an important predisposing factor. The aphthæ begin as small vesicles, and appear in

successive crops. The vesicles soon rupture and leave rounded, shallow ulcers, with yellowish bases and reddish borders. The local symptoms are those of catarrhal stomatitis. The duration of the disease is from one to two weeks.

Treatment consists in relieving the indigestion and using mild antiseptic mouth-washes. A solution of potassium permanganate (3 grains to the ounce) has been especially recommended.

Ulcerative Stomatitis.—This form of stomatitis may develop at any period of life, but it is most common in children between five and ten years of age. It is seldom seen except in debilitated subjects. As it is sometimes epidemic, some authorities regard it as contagious. The bacillus of Vincent has been found in a number of cases.

Symptoms.—The alveolar margin of the jaw is chiefly affected. The gum becomes red, swollen, and spongy. A linear ulcer, with a gray, sloughing base, soon forms, and may extend to the cheek. The glands under the jaw are swollen. In severe cases loosening of the teeth and necrosis of the bone may follow. The constitutional disturbance is often marked. Recovery, however, is the rule.

Treatment.—Hygienic conditions must be improved. Potassium chlorate is almost a specific. The dose for a child of three years is from 1 to 3 grains, in dilute solution, every three hours. The ulcers may be painted with a solution of silver nitrate (10 grains to the ounce). The official preparation of hydrogen dioxid, diluted with 3 parts of water, makes a good mouth-wash. Tonics are frequently required.

Other ulcerations of the oral mucous membrane may be traced to scurvy, metallic poisoning, infectious diseases, or to violence in cleaning the mouth (Bednar's aphthæ).

Mycotic Stomatitis (Thrush).—This is a contagious disease of the mouth, excited by the *Saccharomyces albicans*, an organism related to the yeast fungus. It is met with in infants during the first two weeks, and in older children who have become exhausted through other diseases.

Symptoms.—The characteristic feature is the occurrence upon the mucous membrane of small milk-white elevations, which, on forcible removal, leave an abraded surface. Symptoms of catarrhal stomatitis are also present. The disease may extend to the pharynx, esophagus, and larynx. Microscopic examination reveals the mycelial threads of the fungus.

Treatment.—Generally, thrush yields to simple measures, such as removing the patches with a soft rag soaked in a solution of sodium bicarbonate (1 dram to 5 ounces) and cleansing the mouth at frequent intervals with a weak solution of borax or of sodium hyposulphite (20 grains to 1 ounce). Gastro-intestinal derangements should receive attention. To prevent reinfection, everything coming in contact with the child's mouth must be thoroughly cleansed.

Gangrenous Stomatitis (Cancrum Oris, Noma).—This comparatively rare disease usually occurs in children whose vitality has been reduced by one of the specific fevers, especially measles or whooping-cough. The spirillum of Vincent, diphtheria bacillus, or pneumococcus may be found in association with pus cocci.

Symptoms.—The cheek is the part usually affected. Externally it is swollen, hard, red, and glazed; internally there is a rapidly spreading gangrenous ulcer. The putrefaction causes an intensely fetid odor. The general symptoms are severe. The mortality is high—50 to 75 per cent. The duration is from one to two weeks, death resulting from exhaustion, bronchopneumonia, or septic diarrhea. If recovery occurs, it is usually with marked deformity from cicatrization.

Treatment.—The diseased tissue should be destroyed under anesthesia with the actual cautery or nitric acid. After the operation the mouth should be cleansed at frequent intervals with a solution of hydrogen dioxid (1:3) or of potassium permanganate (1 per cent.). Nutritious foods and stimulants are urgently needed.

Mercurial Stomatitis.—This condition may follow the continued administration of small doses of mercury at frequent intervals, the exhibition of a single large dose, or even of a single small dose, if there be an idiosyncrasy to the drug. The existence of nephritis and of cachectic states favors its occurrence.

Symptoms.—The early symptoms are a metallic taste, increase in saliva, and soreness when the teeth are brought forcibly together. The later symptoms are extreme salivation, redness, tenderness, and swelling of the gums, edema of the tongue, fetor of breath, enlargement of the submaxillary glands, and, owing to pain, great difficulty in articulation, mastication, and deglutition. In severe cases ulceration of the mucous membrane, loss of teeth, and necrosis of the jaw may result.

Treatment.—The first appearance of symptoms calls for the withdrawal of the mercury and the administration of a saline purge. The mouth may be rinsed every half hour alternately with a solution of potassium chlorate and one of hydrogen dioxid. Atropin (gr. $\frac{1}{120}$) may be given once a day to lessen the flow of saliva, and morphin at night, if necessary, to procure sleep. Iron and other tonics may be required to combat exhaustion.

Gonorrheal stomatitis is occasionally seen in children who have been infected at birth. It is marked by catarrhal inflammation and the formation of a whitish deposit on the tongue, gums, and cheeks. It generally yields readily to applications of silver nitrate (1 to 2 per cent.).

ACUTE TONSILLITIS

Etiology.—Acute tonsillitis occurs at all ages, but it is particularly common in childhood and adolescence.

Exposure to cold and wet usually excites it, and such exposure is very effective when the system is debilitated or the throat is congested from improper use of the voice. In some instances the disease is epidemic and traceable to milk infec-

iton. As a secondary affection it is of frequent occurrence in acute infectious diseases, as scarlet fever, diphtheria, and variola. It is often the precursor of a systemic infection—rheumatism, chorea, septicemia, etc. Streptococci, staphylococci, diphtheria bacilli, or pneumococci may be found in the exudate.

Varieties.—(1) Follicular or lacunar. (2) Phlegmonous (quinsy).

Symptoms.—The chief symptoms are chilliness, headache and backache, high fever (103° – 105° F.), pain in the throat, difficult deglutition and altered nasal voice, salivation, fetor of the breath, and swelling and tenderness behind the angles of the jaw.

In the *follicular* form the tonsils are red and swollen, and present little yellow spots on their surfaces. These spots correspond to collections of desquamated and degenerated epithelial cells in the lacunæ or crypts of the gland. During convalescence the contents of the lacunæ are often expelled in the form of cheesy pellets having a characteristic unpleasant odor.

Phlegmonous tonsillitis may be unilateral or bilateral. The affected glands are extremely swollen, sometimes almost touching one another. The pain is intense, and often radiates to the ear. The secretions of the mouth are increased. Swallowing is difficult or impossible, the voice is muffled, and breathing is embarrassed. In three or four days, however, the swollen gland softens from the formation of an abscess, and usually by the fifth or sixth day the pus is discharged spontaneously, with almost instant relief to the patient.

Complications.—The disease usually terminates in recovery, but acute endocarditis, acute nephritis, arthritis, and even general septicemia may occur. Otitis media and phlebitis of the internal jugular vein occasionally ensue. Chronic tonsillitis is a frequent sequel.

Diagnosis.—Follicular tonsillitis must be distinguished from scarlet fever and diphtheria.

Scarlet Fever.—The early and persistent vomiting, the very frequent pulse, the “strawberry tongue,” and the peculiar punctiform eruption will suggest scarlatina.

Diphtheria.—In this disease there is an ashy-gray membrane, which cannot be readily detached, and which, if removed forcibly, leaves a bleeding surface. The membrane does not remain limited to the tonsils, but soon spreads to the pillars, uvula, and pharynx. In doubtful cases the only criterion is the presence or absence of the Klebs-Löffler bacillus.

Prognosis.—This disease is rarely fatal, and even in severe cases recovery is usually complete within ten days.

Treatment.—The patient should be confined to a warm room, and if there be much fever, to bed. A mild aperient is indicated at the outset. The diet should be light but sustaining. The sucking of ice affords relief. The most reliable internal remedies are the salicylic compounds and sodium benzoate. These should be given in full doses at frequent intervals.

R. Tincturæ aconiti..... ℥xl
 Sodii salicylatis..... ℥iss
 Syrupi aurantii..... f℥i
 Aquæ..... q. s. ad f℥iij.—M.
 SIG.—A dessertspoonful every two hours.

Guaiac is also recommended. A dram of the ammoniated tincture of guaiac may be given in milk every three hours. Febrile symptoms, if pronounced, may be controlled by small doses of acetphenetidine or by a combination of aconite and spirit of nitrous ether. The pain may be so intense as to require the use of opium.

Local Treatment.—Externally, cold applications often afford more relief than fomentations. Antiseptic sprays, such as Dobell's solution (see p. 37) or a solution of hydrogen dioxide (1:4), are of decided benefit. Direct applications to the surface of the glands of finely powdered aspirin, of dry sodium bicarbonate, or of the tincture of ferric chloride are often useful.

R.	Potassii chloratis.....	gr. xx
	Tincturæ ferri chloridi.....	fʒiij
	Glycerini.....	fʒv
	Aquæ.....	fʒij.—M.

Sig.—Use locally.

Scarification, followed by gargling with hot water, is another measure which frequently affords relief.

Pus should be evacuated as soon as its presence can be detected. In the majority of cases it is best to make the incision not in the tonsil itself, but in the soft palate, a little above and to the outer side of the gland.

CHRONIC TONSILLITIS

Etiology.—Chronic tonsillitis usually results from repeated attacks of acute tonsillitis and is most common in childhood and early adult life. Streptococci, staphylococci, diphtheroid organisms or tubercle bacilli may be isolated.

Pathology.—The tonsils are usually, but not invariably, enlarged and present an increase in lymphoid cells and connective tissue in varying proportions. The follicles are often dilated and filled with offensive cheesy particles and firm pressure may also reveal pus. Adenoid growths in the nasopharynx are a frequent association.

Symptoms.—An offensive breath, slight cough, increased secretion of mucus and a pronounced tendency to sore throat are the most constant symptoms. Mouth-breathing, snoring during sleep, a nasal quality in the voice, impairment of hearing, a listless facial expression, mental dulness, night-terrors, and malnutrition may also occur, especially when adenoids coexist. Eventually, owing to interference with breathing, deformity of the chest, either of the rachitic or emphysematous type, may supervene.

Chronic tonsillitis increases the liability to diphtheria and is an important cause of otitis media, cervical adenitis (both simple and tuberculous), rheumatism, arthritis deformans, endocarditis, myocarditis and nephritis.

Treatment.—Complete tonsillectomy is usually advisable, but the operation should not be performed during acute exacerbations. General hygienic measures and the use of iron, arsenic, cod-liver oil, etc. are, as a rule, also indicated.

ACUTE PHARYNGITIS

(Acute "Sore Throat;" Simple Angina)

Definition.—An acute catarrhal inflammation of the mucous membrane of the pharynx, soft palate, and uvula. It is frequently associated with tonsillitis and laryngitis.

Etiology.—Exposure to cold and wet is the most common cause. It may be of rheumatic or gouty origin. It may be excited by local irritants, such as hot drinks or the inhalation of noxious gases.

It is also met with in scarlet fever, measles, and other infectious fevers.

Symptoms.—These comprise chilliness, slight fever with its associated phenomena, stiffness and tenderness of the muscles of the neck, soreness in the throat; painful deglutition, a sensation of dryness or tickling, and a hacking cough. Extension to the larynx may cause hoarseness; to the ear, through the Eustachian tube, deafness. Inspection reveals a red and swollen mucous membrane.

Prognosis.—Favorable.

Treatment.—In mild cases a gargle of potassium chlorate will suffice. In severe cases the application to the throat of cloths wrung out of cold water proves grateful. The sucking of pieces of ice affords much relief. Gargles or sprays of the distillate of hamamelis (50 per cent.) are useful. A spray of menthol, 2 grains to the ounce of liquid petrolatum, is also efficacious. Lozenges containing cocain will often relieve pain and allay the tickling sensation in the throat. The following formula, recommended by Bosworth, answers the purpose admirably:

R̄. Cocainæ hydrochloridi.....	gr. v.
Extracti krameriae.....	gr. ij
Sodii bicarbonatis.....	gr. xv
Extracti glycyrrhizæ.....	℥iiss.—M.
Fiant trochisci No. xxx.	

Internally, a mild aperient may be given at the outset. Sodium benzoate (5 grains four times daily) has a beneficial effect. Belladonna with aconite is also recommended. The rheumatic form usually yields promptly to a mild salicylic preparation such as salophen or aspirin (5 to 8 grains three or four times a day).

VINCENT'S ANGINA

This is a disease affecting the tonsils and uvula, less frequently the mouth and pharynx, or lips, and characterized by the formation of a false membrane and, in marked cases, ulceration extending into the submucosa. The breath is peculiarly fetid and the corresponding lymph glands are enlarged, but the constitutional symptoms are, as a rule, comparatively mild. The average duration is from one to three weeks. Differentiation from diphtheria may be impossible without a bacteriologic examination. The disease is feebly contagious and associated with an actively motile spirillum and a fusiform bacillus. Treatment consists in the local application or tincture of iodine, salvarsan, or copper sulphate.

LUDWIG'S ANGINA

(Angina Ludovici)

This is a very grave and acute form of phlegmonous inflammation of the tissues about the floor of the mouth and sides of the neck. It may occur in the course of various specific fevers, or it may be excited by traumatism or carious processes at the roots of the teeth. It may end in abscess-formation or gangrene, and frequently leads to general septicemia.

CHRONIC PHARYNGITIS

Etiology.—Chronic “sore throat” may result from repeated acute attacks, from overuse or improper use of the voice, or from the prolonged action of irritants, like tobacco-smoke. It is a frequent attendant upon chronic nasal catarrh and indigestion.

Varieties.—(1) Hypertrophic; (2) atrophic.

Symptoms.—The voice is husky, and its use is followed by distress; secretion is increased, so that there is a constant desire to clear the throat; disagreeable sensations, as fulness, tickling, and the like, are frequently noted.

In the hypertrophic form (granular sore throat, clergyman's sore throat, chronic follicular pharyngitis) the mucous membrane is thick, swollen, traversed by dilated veins, and studded with numerous elevations which correspond to distended follicles and overgrown lymphatic tissue.

In the atrophic form (pharyngitis sicca) the mucous membrane is pale, smooth, glossy, and dry.

Treatment.—The removal of the cause is of prime importance. All sources of local irritation, such as misuse and overuse of the voice, mouth-breathing, excessive smoking, and intemperance in eating and drinking, must be avoided. Patients should be instructed to expel sounds by the aid of the diaphragm and abdominal muscles instead of the muscles of the throat. Nasal obstructions and adenoid growths must be removed. The habit of hawking and scraping to clear the throat should be rigidly interdicted. Digestive disturbances should receive careful attention. Tonics, such as iron, strychnin, and cod-liver oil, are sometimes required.

Local Treatment.—The nasopharynx should be kept clean by frequent spraying with an antiseptic alkaline liquid, such as Dobell's solution:

℞. Sodii bicarbonatis	
Sodii boratis.....	āā gr. xv
Acidi carbolici.....	gr. viij
Glycerini.....	f℥ij
Aquæ.....	f℥ viij.—M.

Astringent applications are often of service; one of the following may be employed: Zinc sulphate, 5 grains to the ounce; tannin, 1 dram to the ounce of glycerin; silver nitrate, 10 to 20 grains to the ounce. In the follicular variety it is advisable to destroy the enlarged follicles by means of the

galvanocautery, after which the astringent applications may be made.

RETROPHARYNGEAL ABSCESS

(Retropharyngeal Lymphadenitis)

This is a suppurative inflammation of the pharyngeal lymphatics, usually secondary to one of the specific fevers, to follicular tonsillitis, suppurative rhinitis, otitis media, or to caries of the cervical vertebræ. It occurs especially in children. It may be recognized by pain in the throat, dysphagia, dyspnea, alteration in the voice, and the detection, on inspection or palpation, of a swelling projecting from the posterior pharyngeal wall.

Treatment.—As soon as pus can be detected it should be evacuated by means of a guarded bistoury, the head of the child being held forward to prevent the escape of the pus into the larynx.

STENOSIS OF THE ESOPHAGUS

Varieties.—(1) Functional obstruction due to spasm (esophagismus). (2) Organic obstruction.

SPASM OF THE ESOPHAGUS

(Esophagismus)

Etiology.—Esophageal spasm may occur as a symptom of hydrophobia or hysteria; it may be a reflex condition resulting from gastric ulcer, cholecystitis, etc.; it may depend upon a lesion of the esophagus itself, such as ulcer or carcinoma; or it may occur as a primary disorder in neuropathic individuals. It usually affects the cardiac sphincter (*cardiospasm*).

Symptoms.—The symptoms are difficulty in deglutition, often paroxysmal or periodic, regurgitation of food, and in some cases discomfort or actual pain during the ingestion of food. Dilatation of the esophagus may ensue.

Diagnosis.—The functional nature of the obstruction may be recognized by the marked fluctuation or periodicity in the

symptoms, the slowness with which emaciation appears, the slight or transitory resistance offered to the passage of large-sized bougies, and the results of an x-ray examination.

Treatment.—Any underlying neurotic condition should receive careful attention. Bromids and belladonna are sometimes useful. The systematic passage of bougies is usually followed by satisfactory results.

ORGANIC ESOPHAGEAL OBSTRUCTION

Etiology.—Stenosis of the esophagus may be due to (1) external pressure produced by an aneurysm, a tumor, enlarged glands, etc.; (2) cicatrization of an ulcer produced by corrosive acids or alkalis, syphilis, or the action of the gastric juice (peptic ulcer); (3) carcinoma of the esophageal wall.

Symptoms.—The chief symptom is slowly increasing difficulty in deglutition, with the regurgitation of food. The esophagus is often much dilated above the constriction, and the food may collect in the pouch thus formed, so that regurgitation may be delayed for several hours. The passage of a bougie meets with a persistent obstruction. There is much loss of flesh.

Diagnosis.—The history of syphilis or of corrosive poisoning will suggest a cicatrix. Aneurysmal obstruction can usually be detected by physical examination. Aneurysm should be excluded before a bougie is passed. The age, cachexia, pain, emaciation, expectoration of blood-streaked mucus, and enlargement of the cervical glands will suggest carcinoma. The location and the degree of the stenosis are best determined by radiography or esophagoscopy.

In cicatricial stenosis and in some cases of cancerous stricture gradual dilatation with bougies is indicated. Eventually nutrient enemas and possibly gastrostomy will be demanded.

DIVERTICULA OF THE ESOPHAGUS

Two forms of diverticula—circumscribed pouch-shaped dilatations—are observed: pressure diverticula and traction diverticula. The former result

from pressure within the esophagus (large boluses of food) upon an area that is congenitally weak. The most common location is at the junction of the pharynx and esophagus. Traction diverticula are due to the contraction of scar tissue (inflamed lymph-nodes) attached to the outer wall of the esophagus. Symptoms occur chiefly with pressure diverticula and consist of dysphagia and regurgitation of food. The diagnosis is best made by radiographic examination.

DISEASES OF THE STOMACH

ACUTE GASTRITIS

(Acute Gastric Catarrh)

Etiology.—As a primary condition, acute gastritis may result from the eating of indigestible food, partially decomposed food or excessive quantities of food; or it may follow the ingestion of chemical irritants—alcohol, strong acids or alkalis, corrosive sublimate, etc. As a secondary condition it is present in many of the acute infectious diseases.

Pathology.—The mucous membrane is red, swollen, and covered with thick mucus. It is sometimes the seat of ecchymosis. The microscopic changes consist in marked mucoid degeneration and cloudy swelling of the epithelial cells, and the infiltration of the interstitial tissues with round cells.

In toxic gastritis there is often extensive sloughing of the gastric mucosa.

Symptoms.—The symptoms vary much in degree. In mild cases there are anorexia, a feeling of discomfort and fulness, eructations, nausea, and, perhaps, vomiting. The tongue is heavily coated. In severe cases the symptoms are more marked, particularly the nausea and vomiting. There may be also moderate fever (102° – 103° F.), thirst, herpes, distention of the epigastrium, local tenderness, and considerable prostration. The vomitus is composed at first of sour, fermented food; later, of mucus and bile. Jaundice may follow from the extension of the catarrh to the duodenum and bile-ducts, and diarrhea from its extension to the intestines.

Toxic gastritis is manifested by intense burning pain in the throat, gullet, and stomach, persistent vomiting of food-remnants mixed with blood and mucus, marked abdominal tenderness, and the phenomena of collapse. Atrophy of the mucosa and cicatricial stenosis of the orifices are common sequels in cases that do not prove immediately fatal.

Diagnosis.—It may resemble the onset of *scarlet fever*, but the history of contagion, the “strawberry tongue,” sore throat, very rapid pulse, and eruption will lead to the recognition of the latter.

Prognosis.—Simple acute gastritis usually runs a favorable course, and rarely lasts more than a few days.

Treatment.—If the stomach has not been completely emptied, an emetic, such as warm water or ipecac, should be employed. Locally, a mustard-plaster or a turpentine stupe will aid in relieving distress. As a rule, no food should be given by the mouth until the stomach becomes retentive. Ice, however, may be allowed to quench the thirst. In delicate subjects nutrient enemata will be required. If there is constipation, a mercurial laxative may be given with advantage. Such a combination as the following usually acts favorably:

℞. Hydrargyri chloridi mitis..... gr. j
Bismuthi subnitratis..... gr. xx.—M.

Fiant chartulæ No. vi.

SIG.—One on the tongue every hour, to be followed by a Seidlitz powder, if necessary.

Severe pain, nausea, restlessness, and insomnia are best relieved by opium suppositories. Persistent vomiting may be relieved by bismuth subnitrate (10 grains) combined with creosote ($\frac{1}{2}$ minim), with cocain ($\frac{1}{6}$ grain), or with hydrocyanic acid, as in the following formula:

℞. Bismuthi subnitratis..... ʒiij
Acidi hydrocyanici diluti..... ℥xxxij
Aquæ..... fʒiv.—M.

SIG.—Shake well. A dessertspoonful every three hours.

After a lapse of twenty-four or thirty-six hours it is usually possible to give bland nourishment by the mouth. Barley-water, champagne with soda-water, milk and limewater, peptonized milk, and light broths may be given in small quantities at frequent intervals. The return to solid food should always be carried out very gradually.

The treatment of *toxic gastritis* consists in the immediate neutralization of the poison by chemical antidotes, in the evacuation of the stomach (except in the late stages of poisoning by caustics) by the stomach-pump or emetics, and in the administration of demulcents and opium.

CHRONIC GASTRITIS

Etiology.—It may be excited—(1) By prolonged irritation of the stomach, such as results from errors in diet (excesses in eating and drinking, indigestible food, irregular meals, deficient mastication, etc.), or from the excessive use of alcohol, tobacco, condiments, or purgatives; (2) by passive congestion the result of chronic heart disease or cirrhosis of the liver; (3) by chronic diseases that disturb metabolism, such as tuberculosis, diabetes, chronic renal disease, gout, chlorosis, etc.; (4) by chronic diseases of the stomach itself, such as cancer, ulcer, gastrectasia, etc.

Pathology.—The mucous membrane is of a grayish or slaty color, swollen, and covered with tenacious mucus. The veins are dilated, and there may be ecchymoses. Microscopically, there is a cellular infiltration in the interstitial tissue. The glands are dilated, elongated, and tortuous, and their epithelium is more or less degenerated and detached. The interglandular proliferation may be so pronounced as to cause great thickening of the mucous membrane (*hypertrophic gastritis*), or, on the other hand, the new-formed fibrous tissue may contract to such a degree as to cause extreme thinning of the coats of the stomach and atrophy or complete destruction of the glandular elements (*atrophic gastritis*).

Symptoms.—The *subjective symptoms* are very variable, and, for the most part, not characteristic. The chief phenomena are furring of the tongue, fetor of the breath, anorexia, discomfort in the epigastrium, especially after eating solids, belching, eructations, heartburn, constipation, headache, vertigo, and attacks of palpitation. Nausea and vomiting are not uncommon. The latter may occur before breakfast or at the height of digestion. If it occurs on rising in the morning, the vomit consists of frothy mucus; if it occurs after meals, the vomit is composed of undigested food remnants intimately mixed with more or less ropy mucus. The entire epigastrium may be sensitive to pressure.

Examination of the gastric contents reveals an excessive amount of mucus containing many leukocytes, and usually diminished secretion of hydrochloric acid, but not infrequently the acidity is normal or increased (*gastritis acida*). In uncomplicated cases there is no motor insufficiency.

Chronic gastric catarrh rarely terminates in *atrophic gastritis* (*achylia gastrica*), the most important symptoms of which are paroxysmal pain, more or less persistent vomiting, constipation alternating with diarrhea, emaciation, severe anemia, and absence of free HCl and of digestive ferments from the stomach-contents after a test-breakfast.

Diagnosis.—*Atony of the Stomach.*—In simple atony fluids excite as much distress as solids, vomiting is rare, the secretion of mucus is not increased, the secretion of HCl is not usually decreased, and considerable quantities of undigested food can be recovered from the stomach seven hours after a test-meal.

Hyperchlorhydria.—In this condition the general health is not impaired, the appetite is usually good, there is more or less severe pain shortly after eating, albumins and alkalis relieve the pain, an excess of HCl is found in the stomach-contents, albumin-digestion is good, starch-digestion is retarded, and there is no excess of mucus.

Nervous Dyspepsia.—In this syndrome the severity of the symptoms varies considerably from day to day according to

the mental state of the patient, and is not materially influenced by the quantity or the quality of the food; the general health is not often impaired, the nervous symptoms are very prominent, the secretion of the stomach is usually normal, and there is no excess of mucus.

Peptic Ulcer.—The severe, localized paroxysms of pain shortly after eating, the localized tenderness, hematemesis, hyperacidity, and characteristic *x*-ray findings will serve to distinguish ulcer from catarrh.

Cancer of the Stomach.—The history, rapid course, cachexia, persistent vomiting, hematemesis, palpable tumor, signs of gastrectasis, *x*-ray appearances, and the early absence of free HCl from the gastric juice, with the presence of large quantities of lactic acid and of the Boas-Oppler bacilli, will usually render the diagnosis clear.

Care must be taken to determine whether the catarrh is *primary* or *secondary* to some constitutional or visceral disease.

Prognosis.—The primary forms of chronic gastritis, if not too far advanced, are frequently cured. The prognosis is unfavorable when there is much atrophy of the gastric mucosa. In the secondary forms the prognosis is dependent on that of the primary disease.

Treatment.—The cause should be ascertained and removed if possible. Regularity in the time of meals, slowness in eating, and thorough mastication of food must be insisted upon. The patient should be cautioned against overeating and the taking of large quantities of liquid, especially of iced water, during meals. Overindulgence in alcohol, tobacco, coffee, and tea should be forbidden. The resumption of mental or physical work immediately after meals should also be avoided.

A mixed diet of bland, readily digestible food is required. It may usually include boiled, baked, or grilled beef and mutton, chicken, sweetbread, boiled fish, oysters, soft-boiled or poached eggs, pulled bread, fresh butter, baked potato, young string-beans, small peas, spinach, hearts of celery,

thoroughly cooked cereals, calf's-foot jelly, and junket. Tea, coffee, and cocoa may or may not be permissible.

An exclusive milk diet acts exceedingly well in some cases. Systematic lavage is of great value in severe cases, especially when there is excessive secretion of mucus. When lavage cannot be tolerated, the stomach may be cleansed by a glass of hot alkaline water slowly sipped a half-hour or more before breakfast. The following artificial Carlsbad salt may be used as the alkali:

R̄. Sodii sulphatis..... ℥x
 Sodii bicarbonatis..... ℥iv
 Sodii chloridi..... ℥ij.—M.

SIG.—A teaspoonful in a glass of hot water an hour before breakfast.

In mild cases the administration of a bitter—calumba, gentian, nux vomica—some time before meals often proves efficacious. In many cases an alkali may be added with advantage to the bitter, as in the following formula:

R̄. Sodii bicarbonatis..... ℥iss
 Infusi gentianæ compositi..... f℥vj.—M.

SIG.—A tablespoonful before meals.

When the stomach is highly sensitive, silver nitrate will be found a valuable remedy. It may be given in pill form in combination with hyoscyamus, as in the following formula:

R̄. Argenti nitratis..... gr. vj
 Extracti hyoscyami..... gr. x.—M.

Fiant pilulæ No. xx.

SIG.—One pill a half-hour before meals.

Bismuth subnitrate is also of service in such cases. Diluted hydrochloric acid is sometimes serviceable in replacing the natural acid of the gastric juice. In many cases, however, better results are secured from the administration, during meals, of pancreatin with sodium bicarbonate. Flatulence and fermentation may sometimes be controlled by such antiseptics as bismuth salicylate, creosote, bismuth-beta-naphthol, etc. The following combination is often of value:

℞. Creosoti..... ℥_{xx}
 Bismuth-beta-naphthol..... gr. c
 Pulveris zingiberis..... gr. xxx.—M.
 Pone in capsulas No. xx.
 SIG.—One after meals.

So far as possible, constipation should be overcome by regulation of diet, systematic exercise, and the use of enemas or suppositories.

Change of scene, a sunny climate, good hours, and freedom from business worry and household cares often prove more beneficial than any other measure employed.

ATONY OF THE STOMACH

(Motor Insufficiency; Myasthenia Gastrica)

Definition.—Atony of the stomach consists in relaxation of the muscular coat of the stomach and insufficiency of its propulsive power. It frequently leads to gastrectasis.

Etiology.—Motor insufficiency is of common occurrence. It may be congenital; it may be caused by intemperance in eating and drinking; it may follow acute infections; it may occur in the course of chronic diseases attended by malnutrition; it may appear acutely after traumatism or intense emotional excitement; it may be a complication in other diseases of the stomach, especially in gastroptosis, chronic gastritis, nervous dyspepsia, and hypersecretion.

Symptoms.—In simple atony the chief symptoms are a feeling of fulness and discomfort after meals, especially if the latter have been large, and frequent belching of gas. The severity of the symptoms often bears a definite relation to the quantity of food taken. Fluids are as likely to excite distress as solids. As a rule, there is neither vomiting nor pain. The appetite is usually good, the general health is not seriously affected, and the symptoms entirely abate upon the evacuation of the stomach. There are no signs of gastrectasia. When the intestines are similarly affected, there may be marked

nervous symptoms—headache, vertigo, and paresthesia—and considerable disturbance of nutrition.

An exact diagnosis may be made by the stomach-tube or the *x*-ray. No remnants of food are obtained from the stomach before breakfast, but a moderate amount of residue may be recovered from the stomach 7 hours after the Riegel test-dinner or may be seen in the stomach 6 hours after a barium meal.

Prognosis.—This is favorable, if the cause can be removed.

Treatment.—The first indication is to remove the cause. The food should be readily digestible, small in bulk, finely divided, and nutritious. Fluids, except in moderate quantities, and coarse vegetables are to be avoided. The diet may include tender meats, eggs, oysters, boiled fish, well-cooked cereals, steamed rice, stale bread, fresh butter, baked potatoes, tender spinach, string-beans, and asparagus-tips. It is rarely necessary to increase the number of meals. Rest for at least an hour after large meals is to be urged. Exercise in the open air and frequent tepid baths are general measures of value. Lavage is unnecessary unless there are gastrectasis and fermentation.

General tonics, especially iron, are often needed. The most useful direct remedies are the bitters (quassia, gentian, and calumba), particularly the tincture of *nux vomica*, which may be given in doses of from 5 to 10 minims, gradually increased, before meals. Alkalis are indicated when there is excessive acidity. Antifermentatives, particularly small doses of creosote, phenol, or betanaphthol, are useful in reducing flatulence.

Constipation is best relieved by diet, abdominal massage, and enemas.

NERVOUS DYSPEPSIA

(Neurasthenia Gastrica)

Definition.—The characteristic feature of this syndrome is pronounced discomfort during the period of digestion,

out of all proportion to the disturbances of gastric secretion or motility. The source of the discomfort appears to be an excessive irritability of the nerves of the stomach.

Etiology.—Nervous dyspepsia usually occurs in those of a distinctly nervous temperament, and mental overexertion, worry, and excesses are potent etiologic factors. It is frequently associated with neurasthenia and hysteria. It may be due to reflex irritation from other organs.

Symptoms.—The tongue is often clean. The appetite is very variable—at one time it is lost, at another it is inordinate, at another it is perverted, the patient craving unusual articles for food. Pain during the period of digestion is a prominent symptom. It varies in intensity from a feeling of discomfort to the most violent distress. There is rarely tenderness, but the skin over the stomach is often abnormally sensitive.

Belching is common. Vomiting is not frequent. Exaggerated peristaltic movements attended with gurgling sounds (peristaltic unrest) may be perceptible to the patient. Nervous phenomena—headache, vertigo, disturbed sleep, hypochondriasis, lassitude, and palpitation—are conspicuous.

Gastric acidity is usually normal, but there may be subacidity or hyperacidity. In the majority of cases gastric motility is not affected, the viscus emptying itself within the normal period.

The symptoms are usually confined to the period of digestion; they are out of proportion to the disturbance of the digestive functions; they vary greatly from day to day, according to the mood of the patient; and they are not materially influenced by the quality or the quantity of the food.

Prognosis.—This is good, if the cause can be removed.

Treatment.—The treatment is largely that of neurasthenia. The avoidance of excitement and of excessive mental work must be enjoined. An extended voyage may effect a cure. In brain-workers systematic exercise in the open air and frequent bathing, followed by friction of the skin, often prove

very efficacious. On the other hand, the exhausted and anemic may demand the "rest-cure." The diet should be bland and readily digestible. In many cases milk is an appropriate food. Tonics, as iron and arsenic, are often indicated. Short courses of an unirritating bromid, as that of strontium, sometimes do good. The following combination of antispasmodics is useful in certain cases:

℞. Zinci valeratis..... gr. xxx
 Extracti sumbul..... gr. xx.
 Arseni trioxidi..... gr. $\frac{1}{3}$
 Extracti gentianæ..... gr. xx.—M.
 Fiant pilulæ No. xx.
 SIG.—One pill after each meal.

HYPERCHLORHYDRIA

(Superacidity; Hyperacidity)

Definition.—These terms are used to designate an abnormal increase in the secretion of hydrochloric acid during the digestive act. Symptoms are not likely to arise from hyperacidity unless there is also excessive irritability of the gastric mucosa.

Etiology.—This anomaly of secretion is most frequently seen in nervous individuals between the ages of fifteen and forty. Mental overexertion, the excessive use of tobacco, overindulgence in condiments, and insufficient mastication are important predisposing factors. It is a common complication in gastric ulcer, chronic appendicitis, abdominal adhesions, chronic cholecystitis, chlorosis, tabes dorsalis, and migraine.

Symptoms.—The symptoms do not appear immediately after eating, but at the acme of digestion, and include sensory irritation, varying in degree from slight discomfort to agonizing pain, with acid eructations, heartburn, thirst, diffuse tenderness over the stomach, and occasionally vomiting. These symptoms are relieved by eating small quantities of albuminous food and by the ingestion of alkalis, and

disappear spontaneously upon evacuation of the stomach. They may be continuous or periodic. Persistent interdigestive or fasting hyperchlorhydria is strongly suggestive of ulcer.

The stomach-contents obtained after a test-breakfast are excessively rich in hydrochloric acid, both free and combined. Albumin digestion is rapid. The resting stomach is empty. There is no motor insufficiency.

Prognosis.—In the absence of complications the prognosis is favorable.

Treatment.—The cause should be ascertained and removed, if possible. Thorough mastication is imperative. The diet should be unirritating, and composed of the more digestible meats, farinaceous foods, and vegetables. Coarse substances, vinegar, spices, condiments, coffee, and alcohol should be avoided. A moderate amount of water or weak tea at meals is desirable. Fats, in the form of cream and butter, are usually well borne. It is sometimes desirable to increase the number of meals.

Alkalis, in the form of sodium bicarbonate or magnesia, administered at the height of digestion, relieve the symptoms. Silver nitrate ($\frac{1}{4}$ grain) with extract of hyoscyamus ($\frac{1}{2}$ grain) thrice daily, on an empty stomach, is useful. Silver nitrate (1:2000 to 1:1000) may also be given as a stomach-douche with advantage. As there is often marked irritability of the gastric mucosa in these cases, sedatives, such as bromids, valerates, and sumbul, are sometimes of service.

GASTROSUCCORRHEA

(Reichmann's Disease)

Definition.—This is a functional condition characterized by the secretion of large quantities of gastric juice, even when the stomach is empty. It is often associated with hyperchlorhydria. Two forms have been recognized—(1) the continuous and (2) the intermittent.

Etiology.—The causes of gastrosuccorhea are the same as those which excite hyperchlorhydria.

Symptoms.—In the continuous form the symptoms appear regularly, but with varying intensity, and consist in more or less severe pain, both at the acme of digestion and in the night; vomiting of large quantities of yellowish, acid fluid, even when the ingesta are no longer in the stomach; marked thirst; acid eructations; and headache, sometimes of a migrainous type. The ingestion of a small quantity of albuminous food usually relieves the pain. Albumin digestion is good, but starch digestion is retarded. The diagnosis is rendered certain by the finding of from 50 c.c. to 200 c.c. or more of gastric juice, *without any admixture of food*, in the stomach before breakfast, particularly if lavage has been practised the night before.

Complications.—Gastrectasis may result from imperfect digestion of starches or from spasm of the pylorus excited by excessive acidity. Ulcer may coexist. In rare instances tetany develops.

Diagnosis.—In *hyperchlorhydria* the resting stomach is empty and pain does not occur at night. Care must be taken to exclude *locomotor ataxia*, of which intermittent gastrosuccorhea may be an early symptom.

Prognosis.—This is guardedly favorable in uncomplicated cases. Relapses are common.

Treatment.—This is much the same as that for hyperchlorhydria. The painful attacks may be relieved by the administration of alkalis, or, better, by thorough lavage. Belladonna appears to possess some power to reduce gastric secretion.

GASTRALGIA

(Gastrodynia; Neuralgia of the Stomach)

Definition.—Violent paroxysmal gastric pain, occurring independently of any organic disease of the stomach and of any disturbances of secretion or motility. It is a very rare affection.

Etiology.—It is more common in women than in men. Overwork, worry, sexual excesses, abuse of tobacco, reflex irritation, and anemia predispose to it. It may be a symptom of neurasthenia.

Symptoms.—The characteristic features are paroxysms of intense pain, occurring suddenly at irregular intervals, radiating to the chest and back, bearing no definite relation to eating, and lasting from a few minutes to several hours. Vomiting is rare. Pressure over the stomach may relieve the pain, and so may the taking of food. The *x*-ray appearances are normal.

Diagnosis.—Idiopathic gastralgia must be separated from the paroxysmal pain that occurs in gastric ulcer, gastric cancer, hyperchlorhydria, locomotor ataxia, angina pectoris, and in renal and biliary colic.

Gastric Ulcer.—Pain is excited by food and digestion, disappears upon evacuation of the stomach, is associated with hyperacidity, and often with vomiting, hematemesis, and local tenderness.

Gastric Cancer.—The pain is usually more or less continuous, and is aggravated by digestion. There may be persistent vomiting, hematemesis, cachexia, in acidity with lactic-acid fermentation, and a palpable tumor.

Hyperchlorhydria.—The pain is digestive, and is relieved by alkalis and by albuminous food. Examination of the stomach-contents reveals excess of HCl.

Crises of Tabes.—Unsteadiness of gait and of station, Argyll-Robertson pupil, shooting pains in the limbs, abnormalities of sensation, and abolition of deep reflexes will indicate locomotor ataxia.

Angina Pectoris.—The pain radiates from the heart to the neck and arm, is frequently excited by exertion or indiscretions in diet, is generally of short duration, is often attended with immobility of the body and a feeling of imminent dissolution, and is usually associated with the signs of arteriosclerosis.

Renal Colic.—The pain radiates from the kidney into the ureter of the affected side, and concretions or blood may be found in the urine.

Biliary Colic.—The pain is usually in the right hypochondriac region, and is often accompanied by chill, fever, and jaundice. The liver and gall-bladder may be enlarged and tender.

Prognosis.—This is favorable in uncomplicated cases.

Treatment.—*The Attack.*—Hot applications are useful. Galvanization (the anode over the stomach and the cathode near the spinal column) often affords prompt relief. The most generally efficacious remedies are antipyrin (8 grains), brandy (1 to 2 fluidrams), aromatic spirit of ammonia ($\frac{1}{2}$ fluidram), chloroform (2 to 5 minims), and diluted hydrocyanic acid (2 minims). These remedies are most efficacious when given in hot water. Such a combination as the following is frequently successful:

R. Chloroformi..... f $\overline{3}$ iss
 Spiritus ammoniæ aromatici
 Spiritus vini gallici
 Tincturæ cardamomi compositæ..... āā f $\overline{3}$ v.—M.
 Sig.—A teaspoonful in hot water every fifteen or thirty minutes.

In very severe cases it will be necessary to resort to morphin.

The Interval.—The cause must be ascertained, and, if possible, removed. The habits of the patient must be corrected. Methods of treatment intended to improve the general nutrition are of the greatest value. When there is anemia, iron will be found very useful. Among special remedies arsenic, valerianates, sumbul, quinin, and cannabis indica are available. The following combination often proves efficacious:

R. Arseni trioxidi..... gr. $\frac{1}{3}$
 Quininæ valeratis..... gr. xxx
 Ferri pyrophosphatis.....
 Extracti sumbul..... āā gr. xx.—M.
 Fiant in pilulæ No. xx.
 Sig.—One pill after each meal.

In some cases a complete change of scene or enforced rest in bed for a given period is the only means of effecting a cure.

PEPTIC ULCER OF THE STOMACH AND DUODENUM

(Round or Perforating Ulcer of the Stomach and Duodenum)

Definition.—A circumscribed loss of tissue in the stomach or duodenum, usually involving both the mucous membrane and the deeper structures, and characterized clinically by paroxysmal pain, localized tenderness, vomiting, hematemesis, and hyperacidity of the gastric juice.

Similar lesions occasionally occur at the lower end of the esophagus, and after gastro-enterostomy in the jejunum.

Etiology.—Gastric ulcer occurs with about equal frequency in the two sexes, but duodenal ulcer is 4 times more frequent in males than in females. Dietetic errors, general malnutrition, and the existence of chronic disease in the appendix, gall-bladder or colon are predisposing factors. Occasionally, duodenal ulcer is a sequel of an extensive burn of the skin.

Pathogenesis.—It is generally admitted that these ulcers are due to the digestive action of the gastric juice upon an area of local malnutrition. The cause of the malnutrition is obscure. It has been ascribed to thrombosis or embolism, to bacterial infection, to the elimination of toxins by the gastric or duodenal mucosa and to excessive vagal irritability causing spasmodic muscular contraction.

Pathology.—As a rule, the ulcers are single, but they may be multiple. The most frequent seat in the stomach is the posterior wall, in the lesser curvature, near the pylorus; in the duodenum, in the ascending portion about half an inch from the pylorus. They have a punched-out appearance, are round or oval in outline, and, if recent, are funnel-shaped, with the apex toward the serous coat. The edges are usually smooth, rarely ragged. They vary in diameter from a few millimeters to several centimeters, and may extend to the muscularis or even to the serosa.

Symptoms.—Symptoms of indigestion are generally present. The characteristic symptoms are:

1. *Pain.*—This is usually paroxysmal, severe, and localized. It may radiate to the back or sides. In many cases the pain is induced or aggravated by taking food, and continues until the stomach-contents are removed either by vomiting or by spontaneous discharge into the intestine. In other cases, especially of duodenal ulcer, the pain comes on when the stomach is empty, that is, midway between meals or in the early morning hours (hunger pain) and is relieved by bland food. Very often the periods of pain occur at intervals of weeks or months.

2. *Localized Tenderness.*—Two small areas of tenderness can often be elicited, one in front, below the ensiform cartilage, and one behind, a little to the left of the tenth or eleventh dorsal vertebra.

3. *Vomiting.*—This frequently occurs in from one-half hour to two hours after eating. The vomit usually consists of undigested food and acid fluid.

4. *Hematemesis.*—This occurs in more than one-half of all cases. It is the cause of death in about 20 per cent. of all fatal cases of ulcer. The blood is generally fluid and unaltered, but if retained in the stomach for some time, it may have a coffee-ground appearance. In some cases the blood is discharged entirely by the bowel.

Not rarely the bleeding is "occult," or invisible to the naked eye, but may be detected by the benzidin test.

5. *Hyperacidity.*—An increase of HCl is noted after a test-meal in the majority of cases. The acidity, however, may be normal.

X-ray frequently shows in gastric ulcer a projection or niche at the site of the ulcer, an accessory pocket as a result of perforation, and spastic contractions, especially an indentation of the gastric wall directly opposite the ulcer (incisura), and in duodenal ulcer a distortion of the first portion of the duodenum.

In some cases only the symptoms of dyspepsia are present, while in others all symptoms are absent, the disease passing

unrecognized until sudden perforation or profuse hemorrhage occurs. Usually the perforation is into the peritoneum, but very rarely it is into the pleura, pericardium, or intestines.

Sequels.—(1) *Perforation*. This occurs in from 8 to 10 per cent. of all cases. (2) *General or circumscribed peritonitis*. General peritonitis is usually the result of perforation; circumscribed productive peritonitis is a conservative process and results from the direct extension of the inflammatory process through the stomach-wall. It gives rise to perigastric adhesions. (3) *Subphrenic abscesses*. This is usually the result of perforation after the formation of adhesions. (4) *Stenosis of the pylorus, stenosis of the cardia, or hour-glass constriction of the stomach* may result from the contraction of cicatrices. (5) *Cancer* not infrequently develops on the basis of an old ulcer.

Diagnosis.—*Hyperchlorhydria*.—In this condition the pain does not occur so regularly nor so soon after eating; it is not modified by position, but is often completely relieved by eating albuminous food. Hematemesis is absent, there are no tender spots and the *x-ray* findings are negative.

Gastralgia.—In this affection the pain occurs at irregular intervals, is not dependent upon eating (often occurring when the stomach is empty), is relieved by pressure, and is not associated with tender spots, hematemesis, or hyperacidity.

Cancer of the Stomach.—The history, rapid course, advanced cachexia, palpable tumor, vomiting of large quantities of undigested food at long and irregular intervals, "coffee-ground vomit," abundance of lactic acid with Boas-Oppler bacilli, the absence of free hydrochloric acid and the projection of a mass into the barium-filled lumen of the stomach (filling-defect), as shown by *x-ray*, will point to cancer.

Duodenal Ulcer.—In this lesion the pain is further to the right and occurs, as a rule, later after the meals, the blood is usually evacuated through the bowel, and there is little or no vomiting.

Cholelithiasis.—In this condition the pains appear more suddenly, occur at more irregular intervals, and often inde-

pendently of eating, usually radiate toward the right shoulder, and are often associated with swelling and tenderness of the liver, enlargement of the gall-bladder, slight fever, chill, and jaundice.

Prognosis.—This is guardedly favorable in recent cases. The mortality in all cases is from 8 to 10 per cent. Some ulcers run a rapid course and end fatally through hemorrhage or perforation; others, even without treatment, persist for many years. Relapses are common.

Treatment.—Rest and appropriate diet are the most important factors in the treatment. The rest should be kept up for from six to twelve weeks, and for the first two or three weeks of this period the patient should be confined to bed. If hemorrhage has recently occurred or if vomiting be urgent, it is advisable to withhold all food from the stomach for a few days and to nourish the patient by means of nutritive or saline enemas.

After the pain and vomiting have sensibly abated, feeding by the mouth should be resumed. The diet should consist of milk, buttermilk, beef-juice, animal broths, egg-white, and thin pap. As soon as the gastric symptoms have completely disappeared, which will rarely be before the lapse of three or four weeks, the patient may be allowed such articles as soft-boiled eggs, scraped beef, boiled sweet-breads, the tender part of oysters, white meat of chicken, well-made gruel, and custard pudding.

The most useful drugs are the insoluble salts of bismuth (subcarbonate or subnitrate) silver nitrate, and the alkalis. When there is hyperacidity 30 grains of bismuth subcarbonate, alone or with sodium bicarbonate or magnesia, may be given on an empty stomach several times a day. Artificial Carlsbad salt (see p. 46) is an excellent alkaline laxative; of this, a teaspoonful or more may be given in a half pint of hot water in the early morning. Silver nitrate is often of service, and for short periods may be used alternately with bismuth subcarbonate or subnitrate. It may be prescribed as follows:

℞. Argenti nitratis
 Extracti hyoscyami..... āā gr. vj.—M.
 Fiant pilulæ No. xx.
 Sig.—One pill 3 or 4 times a day half an hour before meals

Pain and vomiting usually yield to complete rest, rectal feeding, and the administration of silver nitrate or bismuth subcarbonate. In some cases it may be necessary to use morphin hypodermically. Externally, stupes or sinapisms are sometimes useful. The treatment of hematemesis is considered on page 67.

Surgical Treatment.—In all cases of perforation an operation should be done at the earliest possible moment. If life is threatened by repeated hemorrhage, operation in the interval between the attacks offers the best method of relief. Again, an operation (gastro-enterostomy or partial gastrectomy) is indicated if there is evidence of pyloric obstruction, or of hour-glass contraction or other serious deformity of the stomach, or if the disease does not yield to medical treatment and the life of the patient is endangered by malnutrition.

CARCINOMA OF THE STOMACH

Etiology.—*Sex.*—Carcinoma of the stomach is somewhat more common in men than in women.

Age.—The majority of cases occur between the ages of forty and sixty. It is rare before thirty.

Heredity.—About 8 per cent. of the cases appear to be hereditary.

Prolonged Irritation.—Cancer sometimes develops on the basis of an old ulcer.

Pathology.—Cancer of the stomach is almost always primary. The pylorus is the part most frequently attacked. After the pylorus the points of predilection are the lesser curvature and cardia. The following varieties are encountered: Scirrhus or hard cancer, medullary or soft cancer, adenocarcinoma (cylindric-celled epithelioma), colloid cancer, and squamous-celled epithelioma. Ulceration is rare in scir-

thus, but common in medullary cancer and adenocarcinoma. Colloid cancer appears most commonly as a diffuse infiltration of the stomach-wall. Squamous-celled cancer is rare, and occurs only at the cardia.

Owing to stenosis of the pylorus the stomach is usually dilated. Stagnation of the stomach-contents and the absence of hydrochloric-acid secretion favor the development of lactic-acid fermentation.

Symptoms.—Symptoms of dyspepsia are usually present. The characteristic phenomena are:

1. *Pain.*—This is rarely intense; though aggravated by eating, it is often more or less continuous. It may radiate to the back.

2. *Vomiting.*—This is very common. When the pylorus is obstructed, the vomiting is persistent and occurs long after eating, sometimes at intervals of several days. The vomit is frequently large in amount, and is composed chiefly of undigested food and turbid fluid. It very rarely contains sarcinæ, but long, thread-like bacilli (*Boas-Oppler bacilli*) are almost constantly present and possess some diagnostic significance.

3. *Hematemesis.*—As the bleeding is slight and the blood remains for some time in the stomach, the vomit in many cases acquires a coffee-ground appearance. For a long time the bleeding may be "occult."

4. *Enterorrhagia.*—Blood in the stools, often only recognizable by chemical tests, is present in a large percentage of cases.

5. *Cachexia.*—The anemia, weakness, and emaciation are often disproportionate to the loss of nourishment.

6. *Palpable Tumor.*—A movable, tender mass can be detected sooner or later in a large proportion of all cases.

7. *Absence of HCl with Lactic-acid Fermentation.*—The absence of free HCl and the presence of large quantities of lactic acid, while not peculiar to cancer, are confirmatory indications of the disease.

In addition to these features the symptoms of *gastrectasis* are frequently present and the x-ray commonly shows the projection of a mass into the barium-filled lumen of the stomach (*filling-defect*) and *delayed emptying of the stomach*.

Complications and Sequels.—Metastases in neighboring structures—liver, lymph-glands, pancreas, and peritoneum—are of common occurrence. Ascites and edema are occasionally encountered. Perforation, subphrenic abscess, tetany, venous thrombosis, multiple neuritis, and coma (from acidosis) are rare complications.

Diagnosis.—The differential points between cancer and ulcer and cancer and chronic gastritis have already been considered.

Prognosis.—The disease is almost invariably fatal. The average duration of life is from one to two years. Marked temporary improvement frequently occurs under treatment and may prove very misleading.

Treatment.—In the early stages of the disease, when the pylorus is still patulous, a mixed diet of readily digested food is often well borne. Later, when there is retention, food should be selected that will make small demands on the stomach and that will leave little residue. Bitters—calumba, gentian, condurango—are sometimes employed with advantage. In many cases, but by no means invariably, hydrochloric acid and pepsin are useful. Lavage affords the best means of relieving the distressing symptoms resulting from retention. Vomiting not dependent upon retention may be treated with such remedies as carbonated water, hydrocyanic acid, creosote, cerium oxalate, and bismuth subnitrate. In obstinate cases rectal feeding may be required for a time. Acid eructations and flatulency are sometimes relieved by antacids and internal antiseptics, but generally lavage is much more effective. Pain will require opium, sedatives such as hydrocyanic acid or chloroform, and hot applications.

Early operative intervention may prolong life for several months or several years and rarely effects a cure.

DILATATION OF THE STOMACH

(Gastrectasis)

Etiology.—Chronic gastrectasis may result from—(1) Atony of the stomach-walls (see p. 47); (2) from stenosis of the pylorus.

Stenosis of the pylorus may be caused by—(a) Congenital stricture; (b) carcinoma of the pylorus; (c) cicatrix from ulcer; (d) hypertrophy of the pylorus from gastric catarrh or pylorospasm excited by hypersecretion; (e) pressure from without, as by tumors, adhesions, floating kidney, etc.

Pathology.—All degrees of dilatation are encountered. The most severe forms are usually the result of pyloric stenosis. In atonic dilatation the stomach-walls are thin and atrophic; in dilatation from obstruction there may be marked muscular hypertrophy at the pyloric end.

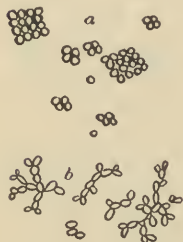


FIG. 1.—a, *Sarcina ventriculi*. b, *Torula cerevisiae*.

Symptoms.—These vary with the cause and the degree of dilatation. In well-marked cases the chief symptoms are a feeling of fulness and discomfort after meals, frequent belching and acid eructations, increased thirst, constipation, deficient urination, and more or less emaciation. Owing to reflex irritation or autointoxication nervous symptoms often develop.

Vomiting is a characteristic symptom, especially when there is stenosis of the pylorus. It occurs long after meals, sometimes at intervals of several days. The vomit is often excessive in amount, is sour and fermented, and on standing separates into a sediment of undigested food and a supernatant liquid, which is turbid and frothy. Not infrequently the vomit contains remnants of food that was eaten several days before. Microscopic examination may reveal, in atonic dilatation, numerous yeast-cells and sarcinae, and in cancerous dilatation, the thread-like bacilli of Oppler.

PHYSICAL SIGNS.—*Inspection.*—The abdomen may be unduly prominent. In some cases the outlines of the enlarged stomach are distinctly visible. Peristaltic waves are frequently seen, especially in stenotic dilatation.

Palpation.—In many cases of obstructive dilatation a tumor can be felt at the pylorus.

Percussion.—After artificial inflation of the stomach with air or carbonic-acid gas percussion reveals an increased area of gastric tympany, but the information afforded by this means is far from being reliable.

Auscultation.—The detection of splashing sounds over the stomach in the morning before breakfast or seven hours after a Riegel test-meal is suggestive of dilatation.

Roentgen-ray.—Examination of the stomach with the roentgen-ray after a meal containing an opaque salt yields trustworthy information as to the exact size of the stomach.

Examination of the Stomach-contents.—The detection of remnants of food in the stomach in the morning after a simple supper renders the diagnosis of severe motor insufficiency certain, and that of obstructive dilatation highly probable.

Complications.—Gastroptosis is a common complication; it may be secondary to the dilatation or the cause of it. Tetany is a rare, but serious, complication.

Diagnosis.—*Gastroptosis.*—In uncomplicated gastroptosis the position of the stomach is lower down than normal and more or less vertical, but there are no signs of motor insufficiency.

Type of Dilatation.—A rapid onset, persistent vomiting, pain, a high degree of motor insufficiency with marked dilatation, a tardy inflow and a rapid outflow of water during lavage, active gastric peristalsis, a palpable tumor at the pylorus, and rapid emaciation suggest pyloric obstruction.

Prognosis.—This depends upon the cause, degree, and duration of the dilatation. In pyloric obstruction the prognosis is grave, although operation may lead to cure, especially in benign forms. Even in atonic dilatation the prognosis

must be guarded if the process is advanced and there is pronounced motor insufficiency.

Treatment.—The food should be nutritious, small in bulk, and readily digestible, and, in advanced cases, should be given in small amounts at frequent intervals. Liquids should never be given in large quantities. In severe grades of dilatation, to prevent the tissues from losing water, it is advisable to introduce fluids in the form of water and meat-broth by the bowel. To prevent retention, to control fermentation, and to cleanse the stomach, no measure is so useful as methodic lavage. When there is considerable retention, the lavage should be performed daily, preferably in the early morning. A carefully adjusted abdominal bandage nearly always affords comfort and gives mechanical support to the stomach. In cases due to atony exercise in the open air, hydrotherapy, and, unless there be marked gaseous fermentation, abdominal massage are valuable aids. Faradization of the stomach may also be used to promote muscular contraction. In dilatation from muscular relaxation *nux vomica* is very useful. Such remedies as creosote, salol, and bismuth-beta-naphthol are sometimes of service in checking fermentation, but they are much less efficacious than systematic lavage. Constipation is best treated by simple enemas or by glycerin suppositories.

Surgical Treatment.—In the large majority of cases of non-obstructive dilatation medical treatment suffices. Occasionally, however, surgical intervention is demanded on account of persistent suffering and progressive emaciation. The operation indicated in these cases is gastro-enterostomy. In cases of pyloric obstruction of a benign character an operation is indicated when it is impossible to maintain nutrition by proper medical treatment. Two operations are available: pyloroplasty and gastro-enterostomy. The treatment of pyloric cancer is considered on page 61.

Acute dilatation of the stomach is the result of paresis of the gastric muscle, the primary cause of which may be

traumatism, an abdominal operation, or some debilitating disease, notably pneumonia or typhoid fever. The diagnostic features are acute epigastric distress, abdominal distention, persistent vomiting of large quantities of bilious fluid, a dull area on percussion in the left side of the abdomen, great thirst, and progressive collapse. Unless treatment is soon instituted death usually occurs within a few hours or days. The knee-and-elbow posture, thorough lavage, saline enemas, and subcutaneous injections of pituitary extract (15 min.), physostigmin sulphate ($\frac{1}{50}$ grain), and strychnin sulphate are the measures most likely to afford relief.

HYPERTROPHIC PYLORIC STENOSIS OF INFANTS

The stenosis, which is probably congenital and manifests itself within the first 2 or 3 weeks, may be the result of muscular spasm or hypertrophy or of both conditions. The diagnostic symptoms are persistent vomiting, often projectile, retention of milk after a three-hour interval, marked constipation, visible gastric peristalsis, and a palpable tumor at the pylorus. Unless relief is afforded within a week by the use of breast milk or well diluted cow's milk in small amounts at frequent intervals, lavage once or twice daily, enteroclysis with saline solution and the administration of belladonna and bromids, operation (linear incision of serous and muscular coats of pylorus, left unsutured) should be performed.

GASTROPTOSIS AND ENTEROPTOSIS

(Glénard's Disease)

Definition.—Prolapse of the stomach and transverse colon caused by congenital or acquired weakness of the abdominal muscles and ligaments.

Etiology.—The condition is much more common in women than in men. Tight lacing, repeated pregnancies, abnormal formation of the thorax, enlargement of other abdominal organs, gastrectasis, and constitutional weakness are important predisposing factors.

Symptoms.—The chief objective feature is a more or less pronounced downward displacement of the pylorus, in consequence of which the stomach assumes a vertical or

subvertical position. Dislocation of the whole stomach downward is rare. Dilatation of the pyloric extremity is a common sequel. The position and size of the viscus may be determined by percussion after artificial inflation or, much more accurately by an *x*-ray examination after an opaque meal. The transverse colon shares in the downward displacement of the stomach and occupies a position immediately below the greater curvature. Ptosis of other abdominal organs, especially of the right kidney and liver, is also present in many cases. Separation of the recti muscles is often seen. A floating tenth rib is less frequent.

Subjective symptoms are often wanting, but many patients complain of digestive disturbances and not a few become more or less neurasthenic. Occasionally, symptoms of gastrectasis supervene from distortion of the pylorus.

Treatment.—The diet should be adapted to the digestive and motor powers of the stomach. Mechanical support of the pylorus by means of a snugly fitting abdominal bandage affords relief in mild cases. In severe cases, especially if the nervous phenomena are pronounced, a modified rest-cure may prove effective. Lavage is not indicated unless there is general dilatation with retention or excessive secretion of mucus. In very obstinate cases surgical intervention (gastropepy or gastro-enterostomy) should be considered.

HEMATEMESIS

(Gastrorrhagia)

Etiology.—Hemorrhage from the stomach may result from (1) traumatism; (2) gastric ulcer or erosion; (3) gastric cancer; (4) venous engorgement of the stomach following cirrhosis of the liver, or splenic enlargement; (5) acute gastritis; (6) atheroma or embolic or thrombotic obstruction of the gastric vessels; (7) blood dyscrasia, as in scurvy, purpura, severe infections, and grave anemias; (8) rupture of an aneurysm; (9) swallowing of blood from the nose,

mouth, or throat; (10) focal infection in the appendix, gall-bladder, etc.

Symptoms.—The quantity of blood that is vomited varies considerably; rarely a quart or more is lost. In many cases a portion of the blood escapes through the bowel. The blood is usually dark, is often mixed with food, has an acid reaction, and may be fluid or clotted. If the hemorrhage is severe, the symptoms of acute anemia develop—pallor, weakness, vertigo, tinnitus aurium, dimness of sight, syncope, and convulsions.

Diagnosis.—*Hemoptysis.*—The blood is coughed up; it is usually bright red, frothy, and alkaline in reaction; subsequent expectorations are tinged with blood, and the associated symptoms and signs point to pulmonary or cardiac disease.

Prognosis.—Hematemesis is rarely so severe as to cause death. The most dangerous hemorrhages are those that occur in cirrhosis of the liver, splenomegaly, and aneurysm.

Treatment.—In the treatment of hematemesis absolute rest is essential. No food of any kind should be given by the mouth. An ice-bag should be applied over the stomach, and morphin should be given hypodermically. The application of firm bandages to the four extremities may act favorably. Ergot and such drugs as tannic acid, iron sulphate, and lead acetate are of very doubtful utility. A solution of adrenalin chlorid (1:1000) is worthy of trial. A dram in an ounce of water may be given every twenty minutes. If the bleeding is prolonged coagulen (20 min. of a 10 per cent. solution by the mouth) may be tried.

Collapse following hemorrhage will call for diffusible stimulants, the external application of heat, and the subcutaneous or intravenous injection of warm saline solution or the transfusion of blood.

DISEASES OF THE INTESTINES

HABITUAL CONSTIPATION

Definition.—Infrequent or difficult evacuation of the feces.

Etiology.—The chief causes are: (1) Sedentary life, lack of exercise, and persistent neglect of the desire for evacuation. (2) Dietetic errors (food deficient in residue, insufficient water, etc.). (3) Many general diseases that weaken the abdominal muscles, lessen the intestinal secretions or inhibit peristalsis, such as anemia, diabetes, neurasthenia, hysteria, melancholia and organic diseases of the brain and spinal cord. (4) Many chronic diseases of the digestive tract—gastric disorders, enteroptosis, chronic intestinal catarrh and obstructive jaundice. (5) Atony of the intestinal musculature from senility. (6) Mechanical obstruction to the passage of the intestinal contents by abnormal kinks (the result of evolutionary or inflammatory bands), stricture, tumors, torsion, etc. (7) Interference with peristalsis by reflex stimuli produced by various painful conditions of the abdominal or pelvic viscera such as irritable prostate, uterine disease, rectal ulcer, anal fissure, hemorrhoids, etc.

The immediate cause of constipation is usually reduced motility in the lower bowel, but in some instances, as in certain cases of hysteria or neurasthenia or of lesions external to the bowel (chronic appendicitis, cholelithiasis, adhesions, uterine displacements, etc.) it may be spasm of the lower bowel (spastic constipation).

Symptoms.—Some persons continue to enjoy excellent health even though their bowels are evacuated at very infrequent intervals. Usually, however, retention of fecal matter in the intestines longer than is customary with the individual gives rise to unpleasant symptoms, common among which are headache, dizziness, mental sluggishness, lassitude, fetor of the breath, a coated tongue, and anorexia. The diagnosis usually offers no difficulty. Mechanical hindrances are often demonstrable by the x-ray.

Sequels.—Severe persistent constipation may lead to piles, fissure, ulceration of the colon, distention of the colon, diarrhea from irritation, fecal impaction, hernia. Straining in constipation may be the immediate cause of apoplexy, attacks of angina pectoris, or the rupture of an aneurysm.

Treatment.—The removal of the cause is a matter of the first importance. Cathartics should be avoided, if possible. In some cases the activity of the bowels is restored by repeated daily attempts at defecation at some special hour. Systematic exercise and cold bathing are of the greatest benefit. Abdominal massage, especially digital kneading in the direction of the colon, is often quite effectual.

Unless the state of digestion offers a contraindication, such laxative articles of food as green vegetables, oatmeal, cornmeal, whole-wheat bread, oils, and cooked fruits should be ordered. Water-drinking should be encouraged. In mild cases a glass of cold water before breakfast may suffice. In constipation arising from a highly refined diet agar (2–6 teaspoonfuls with gruel or cooked fruit) is useful.

General tonics, as iron and strychnin, are sometimes needed. Mineral waters, as Friedrichshall, Hunyadi János, or the milder Saratoga or Bedford waters, are very useful, but possess no special advantages over the saline laxatives (sodium phosphate or Rochelle salt), if the latter are taken in small amounts well diluted. Enemas of soapy water or of glycerin or suppositories of gluten, soap, or glycerin, often prove highly satisfactory. In obstinate atony systematic

colonic irrigation with cold water is sometimes efficacious. In cases of fecal impaction enemas of warm oil (4-6 ounces, at night) will be found useful. Vegetable cathartics are usually necessary in obstinate cases. The mild ones should always be tried first, and even with these considerable care should be exercised lest the patient comes to rely upon drugs to the exclusion of the hygienic and dietetic measures already indicated. Of the mild laxatives, cascara sagrada is one of the best: from 10 to 30 minims of the fluid extract, or a corresponding dose of a pleasant elixir, may be administered at bedtime and repeated, if necessary, in the morning. In constipation resulting from constrictions of a spastic or an organic nature, liquid paraffin (1-2 tablespoonfuls morning and night, on an empty stomach, the dose being gradually reduced) often affords relief.

In many cases a combination of several laxatives (rhubarb, aloes, podophyllum, euonymin, and colocynth) acts better than any one singly. As adjuvants, nux vomica or physostigma may be added to overcome intestinal atony, and belladonna or hyoscyamus to prevent gripping. The most suitable combination must be determined in each case by experience. A pill, like one of the following, will generally prove satisfactory:

R̄. Aloini..... gr. iv
 Extracti nucis vomicæ..... gr. iv
 Extracti belladonnæ..... gr. iij.—M.

Fiant pilulæ No. xxiv.

Sig.—One pill at bedtime

R̄. Pulveris rhei
 Extracti cascariæ sagradæ..... āā gr. xxiv
 Extracti physostigmatis
 Extracti belladonnæ..... āā gr. iv.—M.

Fiant pilulæ No. xxiv.

Sig.—One pill at bedtime.

In extreme cases suspension of the colon, partial colectomy, or a short circuiting operation may be considered.

INTESTINAL COLIC

(Enteralgia; Tormina)

Definition.—Intestinal pain of a spasmodic character.

Etiology.—It usually results from irritating food, flatulence, or fecal accumulation. It is a common symptom of structural lesions of the bowel—enteritis, dysentery, appendicitis, intestinal obstruction. It is an important symptom in chronic lead-poisoning. It may be reflex from disease of the ovaries, uterus, liver, vertebræ, etc. It may occur as a crisis of locomotor ataxia.

Symptoms.—The chief feature is paroxysmal pain of a cramp-like character, centering around the umbilicus, and relieved by pressure. The abdomen is usually distended. Severe attacks may lead to collapse, indicated by cold sweats, pinched features, feeble pulse, and vomiting. The attack lasts from a few minutes to several hours, and usually ends with a discharge of flatus.

Diagnosis.—*Lead Colic.*—This may be recognized by the history, blue line on the gums, retracted abdominal wall, wrist-drop, and lead in the urine.

Biliary Colic.—Pain radiating from the liver to the back, jaundice, enlargement and tenderness of the gall-bladder, and calculus in the stool are characteristic of gall-stone colic.

Renal Colic.—This is indicated by pain extending from the kidney, along the ureter to the penis and testicle, frequent micturition, blood or calculus in the urine.

Chronic Appendicitis.—Localized tenderness (McBurney's point), muscular rigidity, and induration point to appendicitis.

Intestinal Obstruction.—In this condition vomiting is incessant and often of a stercoraceous character; constipation is absolute, without discharge of feces or flatus, tumor is sometimes present, and collapse symptoms are marked.

Acute peritonitis is revealed by continuous pain, fever, abdominal tenderness, rigidity, and leukocytosis.

Treatment.—The indications are to relieve pain and to remove the cause. Turpentine stupes are useful. In severe cases it will be necessary to give morphin ($\frac{1}{8}$ to $\frac{1}{4}$ grain) and atropin ($\frac{1}{100}$ grain) hypodermically. Carminatives—peppermint, ginger, oil of cloves, Hoffman's anodyne—often afford relief.

Colic excited by irritating food or fecal accumulation is promptly relieved by saline or mercurial purges.

DIARRHEA

Definition.—A condition in which the stools are too frequent and too liquid. Like dyspepsia, it is a symptom of many pathologic conditions.

Etiology.—(1) It is sometimes due to excessive ingestion of water or of certain foods (fats, fruits, coarse vegetables). (2) It is sometimes produced by diseases of the stomach, as incontinence of the pylorus and achylia gastrica. (3) It results from inflammation of the intestines—enteritis, ileocolitis, dysentery (inflammatory diarrhea). (4) It is a symptom of certain infectious diseases, such as typhoid fever and cholera (symptomatic diarrhea). (5) It may be excited by cathartic drugs. (6) It often occurs as a final symptom in cachectic states, as in cancer, diabetes, and chronic renal disease (colliquative diarrhea). (7) It sometimes marks the crisis of acute infections, such as typhus fever and pneumonia (critical diarrhea). (8) It may result from certain nervous influences (emotional excitement, Graves' disease, neurasthenia).

ACUTE ENTERITIS

Etiology.—The occurrence of acute catarrhal inflammation of the intestinal mucous membrane is favored by hot weather, bad hygienic surroundings, and improper food. Chilling of the surface may also be a factor. The direct cause is usually some irritant produced in food through the action of bacteria. Less frequently bacteria themselves are the exciting factors,

The *Bacillus coli communis*, *Bacillus enteritidis*, paracolon bacillus, *Bacillus dysenteriae* (Shiga) or allied organisms, and, less frequently, the *Bacillus proteus*, streptococci or staphylococci, may be the active agents. Acute enteritis may also be excited by mineral poisons (antimony, arsenic, mercury) and by certain autogenic poisons (uremia). As a secondary process it is observed in many infections, as typhoid fever, cholera, dysentery, etc.

Pathology.—The mucous membrane is swollen, injected, and covered with mucus. The lymph follicles are enlarged and not rarely show erosion or actual ulceration. In severe cases small hemorrhages occur and exceptionally the mucous membrane is covered with a more or less extensive false membrane.

Symptoms.—The stools are frequent—three or four to twenty or more a day—watery, brownish, and, as a rule, more or less offensive. The amount of visible mucus is rarely considerable unless the large bowel is especially affected. Undigested food may be present if the upper part of the small bowel is chiefly involved. Accompanying the diarrhea there are usually colicky pains, abdominal soreness, tympanites, borborygmus, and more or less fever, although the temperature may be normal. Nausea and vomiting may be marked if the stomach is also affected, and sometimes jaundice develops from the extension of the inflammation from the duodenum into the bile-ducts. Tenesmus is absent unless the inflammation extends to the lower colon.

Cholera Morbus (Cholera Nostras).—This is a very severe form of acute enteritis usually traceable to food poisoning. It is characterized by persistent bilious vomiting, frequent and copious watery stools, violent abdominal pains of a cramp-like character, marked pyrexia, great prostration and, in extreme cases, collapse. Death rarely occurs, however, except in aged or debilitated subjects.

Diarrhea in Childhood.—The predisposing factors comprise warm weather, improper food, bad hygienic conditions,

dentition and malnutrition. Qualitative changes in the food, especially in milk, the result of bacterial action, is the most common exciting cause. Three types are usually recognized: (1) Simple or dyspeptic diarrhea; (2) ileocolitis; (3) cholera infantum. The line between these types, however, cannot be sharply drawn.

Simple or Dyspeptic Diarrhea.—In this form the stools are offensive and usually contain undigested curds of milk, but little or no visible mucus. Vomiting frequently occurs. The temperature generally ranges from 100° to 103° F. Unless the disease is long continued prostration and emaciation are not marked. Recovery is the rule, although relapses are common and the condition may lead to ileocolitis.

Ileocolitis.—In this form of diarrhea the symptoms are more intense. The stools are numerous and contain much mucus and not rarely streaks of blood. The abdomen is distended and tender. The temperature usually ranges from 103° to 105° F. Vomiting often occurs, but usually it is a less conspicuous feature, than in the dyspeptic form. Colicky pains may precede the stools and not infrequently there is pronounced tenesmus. The *Bacillus dysenteriae* is found in some cases. Exhaustion and emaciation rapidly ensue, and in severe cases the child sinks into a state of complete collapse or passes into a marantic state persisting several days. Death is frequently preceded by extreme apathy, stupor or even coma (spurious hydrocephalus). Bronchopneumonia and acute nephritis are not uncommon complications. The disease is always a serious one, especially in young infants, and even in favorable cases convalescence is likely to be tedious.

Cholera Infantum.—This is the rarest but the gravest form of summer diarrhea. The onset is usually sudden. Vomiting and purging begin almost simultaneously and become incessant. The stools are large and watery. Thirst is intense. The surface temperature is low but the rectal temperature is very high (105°–106° F.). The urine is scanty or suppressed. Collapse soon follows, and is shown by pinched features, hollow

eyes, sunken fontanel, pallid skin and cold surface. Even at this time a reaction may set in, but in the large majority of cases death results in from twenty-four to forty-eight hours. Toward the end stupor, convulsions, and coma (spurious hydrocephalus) may supervene.

Diagnosis.—The diagnosis of acute enteritis usually offers no great difficulty. The gradual onset, nose-bleed, splenic enlargement, continued fever, general prostration, and serum reaction will lead to the recognition of *typhoid fever*. *Peritonitis* may be distinguished by the more intense pain and tenderness, greater tympanites, constipation, and marked constitutional disturbance. As to the location of the inflammation, especial *involvement of the small bowel* is suggested by vomiting, borborygmus, and the occurrence of large fecal stools containing particles of undigested food but little or no visible mucus; *colitis* is suggested by tenderness along the course of the colon, tenesmus, and the occurrence of blood or of large amounts of mucus in the evacuations.

Treatment.—Rest in bed and the substitution of bland nourishment for the ordinary diet are all that is required in many cases. Boiled milk, milk and arrow-root, and mutton, veal, or chicken broth are suitable foods. In robust subjects it may be advisable to withhold all food for twenty-four or thirty-six hours. If the patient is seen at the outset and there is reason to believe that irritant material is still present in the bowel, an unirritating purgative, such as castor oil, Epsom salts, or calomel, should be given. Occasionally a second dose of the purgative may be given with benefit. Externally, sinapisms or stupes are frequently efficacious. If the diarrhea continues mild astringents (bismuth subnitrate or chalk) and opium are indicated. They may often be combined advantageously with antiseptics as in the following formulas:

℞. Morphinae sulphatis..... gr. j
 Phenylis salicylatis..... gr. xxiv
 Bismuthi subnitratis..... ʒss.—M.

Fiant chartulæ No. xii.

SIG.—One every three hours.

R.	Bismuthi subsalicylatis.....	ʒiss
	Cretæ præparatæ.....	ʒiiss
	Tincturæ opii camphoratæ.....	fʒj
	Pulveris acaciæ.....	q. s.
	Aquæ cinnamomi.....	q. s. ad fʒvi.—M.

SIG.—A tablespoonful every three hours.

In *cholera morbus* the hypodermic injection of morphin ($\frac{1}{4}$ grain) and atropin ($\frac{1}{100}$ grain) is necessary. Hot applications to the abdomen are useful. Calomel in fractional doses serves to allay vomiting and to rid the bowel of irritating matter. Collapse will require hot bathing, diffusible stimulants (ammonia, brandy, camphorated oil), and in some instances subcutaneous injections of salt solution.

Acute Enteritis in Infants.—The first indication is to withdraw the milk at once, and to withhold it for several days or until the stools become quite natural. Indeed, in many cases it is well to suspend all nourishment for the first twenty-four hours, allowing nothing by the mouth but barley-water or plain boiled water. Subsequently, albumin-water, fresh beef-juice, veal broth, or a liquid peptone preparation may be given in lieu of milk. Milk feeding should always be resumed very gradually. Absolute rest in the recumbent position is essential. Removal to the seashore or mountains is often of the greatest benefit.

To remove irritant matter from the bowel, castor oil or calomel should be given, preferably the latter when the stomach is sensitive. In most cases it is necessary to follow the purge with a sedative astringent such as bismuth subnitrate or chalk. From 5 to 10 grains of one of these drugs may be given every two or three hours with an intestinal antiseptic (salol, beta-naphthol-bismuth, bismuth salicylate); some such combination as the following may be ordered:

R.	Bismuthi subnitratis.....	ʒij-iv
	Phenylis salicylatis.....	gr. xxiv
	Misturæ cretæ.....	fʒiij.—M.

SIG.—A teaspoonful every two hours.

A more active astringent, such as tannalbin or tannigen (2 to 3 grains), may be given in addition to the bismuth subnitrate or chalk when the discharges are exceedingly profuse and watery.

Opium is often of great value, but extreme caution must be exercised in its use. It is called for when the diarrhea continues in spite of the thorough unloading of the bowel and the administration of mild astringents. From 3 to 5 minims of paregoric may be given every two, three, or four hours, according to circumstances. If the stomach is unretentive, laudanum (1 to 2 minims) may be given by enema.

In *ileocolitis* intestinal irrigation is an important part of the treatment. Once or twice a day the colon should be thoroughly flushed with saline solution or with water containing a dram of sodium benzoate to the pint. After the irrigation an enema of thin mucilage (2 fluidounces) and bismuth subnitrate (2 drams) may be given every three or four hours. If colitis persists, injections of silver nitrate (1:10,000) may be employed.

In *cholera infantum* the stomach should be washed out with warm water, and the bowel irrigated with cold water. At first nothing should be given by the mouth except sterilized ice-cold water and iced brandy or champagne. When the stomach is wholly unretentive, stimulants should be given hypodermically. Hot packs (101.4° F.) are very useful in combating collapse. In urgent cases normal salt solution (40 grains to the pint) should be used subcutaneously, from 2 to 3 ounces being injected three or four times daily. If vomiting and purging still continue, small doses of morphin and atropin should be administered hypodermically. Holt gives $\frac{1}{100}$ grain of morphin with $\frac{1}{800}$ grain of atropin for a child one year old, and repeats the dose, if necessary.

After vomiting has ceased, barley-water, albumin-water, and fresh beef-juice may be given by the mouth. Milk feeding should always be resumed very gradually.

CHRONIC ENTERITIS

Etiology.—Chronic enteritis may follow an acute attack or repeated attacks of acute enteritis, or it may develop gradually as a result of passive congestion of the bowel (chronic heart or liver disease), of faulty digestion, of chronic constipation, or of other conditions of the bowel (carcinoma, stricture, etc.).

Symptoms.—There may be persistent diarrhea or persistent constipation, or alternation of periods of diarrhea with periods of constipation. When the large bowel is especially affected considerable mucus is secreted. In many cases anemia, emaciation, and various nervous symptoms (depression, hypochondriasis, and neurasthenia) are observed.

Diagnosis.—It is important to exclude specific forms of enteritis—tuberculous, dysenteric, and syphilitic. In doubtful cases, a digital examination supplemented, if necessary, by proctoscopy, should be made in order to exclude carcinoma of the rectum.

Prognosis.—The prognosis is uncertain. If the cause can be removed, however, a complete cure may be effected.

Treatment.—The cause must be ascertained and removed, if possible. The diet, clothing, habits, occupation, and mode of living of the patient should receive careful attention. No definite rules can be laid down in reference to the diet. When the disease is not very severe and is confined for the most part to the colon, a selected mixed diet may be allowed. Many patients do well upon an exclusive milk diet. Foods that are bulky and leave much residue are always inadmissible.

Protection of the body against chilling is of vital importance. Woolens should be worn next to the skin. A snugly fitting abdominal bandage may be worn as an additional safeguard. Rest in bed is sometimes essential. When the general nutrition is not too much impaired, a change of air and scene may prove very beneficial.

Mineral astringents, especially bismuth subnitrate (30 to 40 grains), silver nitrate ($\frac{1}{4}$ to $\frac{1}{2}$ grain), copper sulphate ($\frac{1}{4}$ to 1 grain), and lead acetate (1 to 3 grains) are of service.

Intestinal antiseptics—salol, bismuth salicylate, beta-naphthol-bismuth—are useful adjuvants. Opium is often required in acute exacerbations. When the disease is situated chiefly in the colon, irrigation of the bowel two or three times a week with a solution of silver nitrate (10 to 20 grains to 1 pint) is especially to be recommended.

MUCOUS COLITIS

Definition.—Mucous colitis, or mucous colic, is a chronic secretory neurosis of the colon, characterized by the regular or periodic passage of masses of mucus and paroxysms of abdominal pain.

Etiology.—The disease is most common in middle life, and affects women much more frequently than men. It is met with particularly in nervous, neurasthenic, or hysterical individuals, who suffer also from indigestion and constipation. In some cases it is secondary to organic disease of the bowel, as chronic appendicitis, tumor, or stricture.

Symptoms.—The chief phenomena are constipation, the passage of excessive quantities of mucus in the form of flakes, shreds or casts of the bowel, indigestion, and paroxysmal attacks of intestinal colic. Symptoms of hypochondriasis, neurasthenia, or hysteria are also present in greater or less degree in most cases. Emaciation is sometimes marked.

Prognosis.—The disease is very obstinate, but rarely fatal. Marked improvement or even recovery may follow treatment.

Treatment.—The diet should be liberal, solid rather than liquid, and unirritating. The general treatment laid down for neurasthenia or hysteria is often applicable. Mild laxatives, preferably frequent doses of castor oil, should be given. In some cases, however, liquid petrolatum is more effective than castor oil. Colonic douches, several times a week with a solution of sodium bicarbonate (1 dram to 1 quart), or high

injections of warm oil often afford much relief. Short courses of bromids and belladonna may be of service. Any associated abdominal lesion should receive appropriate treatment.

SPRUE

Sprue, or psilosis, is a chronic inflammatory disease of the alimentary canal, occurring in certain tropical countries, and characterized by aphthous stomatitis and glossitis, dyspeptic symptoms, intermittent diarrhea, anemia and emaciation. The stools are characteristically voluminous, frothy, fatty and offensive. Impaired vitality from any cause and an ill-balanced diet seem to be predisposing factors. The exciting cause is not definitely known, but certain authors believe it to be a yeast-like organism—*Monilia psilosis* (*Monilia albicans*). Except in the early stages the outlook is uncertain. Rest, change of residence, a preliminary dose of castor oil, a diet consisting at first solely of milk and later chiefly of meat, eggs and fresh fruits, and the administration of pancreatin are the measures that are most likely to be of service.

ACUTE APPENDICITIS

Definition.—An inflammation of the appendix vermiformis.

Pathology.—There are three varieties: Catarrhal, ulcerative, and interstitial.

Catarrhal Appendicitis.—In mild cases the appearances are, no doubt, similar to those observed in catarrh elsewhere, but in severe cases the wall of the appendix is infiltrated with round-cells, and the mucous membrane is denuded of epithelium and presents a granular surface. This latter condition may eventuate in diffuse chronic appendicitis with relapses (*recurrent appendicitis*), or union of the granulating surfaces with complete obliteration (*appendicitis obliterans*).

Ulcerative Appendicitis.—In this type the wall of the appendix is the seat of a more or less localized ulcer. It may be associated with the presence of fecal concretion or a foreign body, or it may be the result of typhoid or tubercular infection.

Interstitial Appendicitis.—In this form mucous, submucous, and muscular coats are swollen, infiltrated, and the seat of necrotic changes, and the peritoneal covering is congested and roughened. Suppuration may ensue, transforming the

appendix into a pus-containing sac, or the walls of the organ may become gangrenous. In either case spontaneous rupture is likely to occur. In other instances a chronic process follows, in which the appendix becomes thickened, indurated, and adherent to adjacent structures.

Appendicitis is always due to the action of pathogenic bacteria, the chief offenders being the *Bacillus coli communis*, *Streptococcus pyogenes*, *Staphylococcus pyogenes aureus*, typhoid bacillus, and tubercle bacillus. Of these, the *Bacillus coli communis*, a natural habitant of the bowel, is most commonly present. Under ordinary conditions it is harmless, but if the circulation of the appendix is interfered with from any cause or the coats of the tube are abraded, infection is likely to arise.

Etiology.—Appendicitis is more common in males than in females. It is most frequent between the fifteenth and thirtieth years. Exposure, errors in diet, intestinal catarrh, traumatism, and the lodgement in the appendix of fecal concretions or foreign bodies predispose to the disease. It may follow some infection, as typhoid fever, influenza, or tuberculosis. It may be induced by twisting of the appendix.

Symptoms.—These comprise: (1) Sudden pain, often general at first, but later most marked in the right iliac region. (2) Circumscribed tenderness, most frequently detected over McBurney's point—a point midway on a line between the umbilicus and the anterior superior iliac spine. (3) Fever, ranging between 100° and 103° F. (4) Localized rigidity in the right iliac fossa, or the presence of a definite tumor. (5) Reflex disturbances—nausea, vomiting, and constipation, or, rarely, diarrhea. (6) Leukocytosis of from 12,000 to 20,000 per c.mm.

Terminations.—Complete subsidence of the inflammation, with the likelihood of other attacks, chronic appendicitis, general peritonitis, and localized abscess. The location of the abscess depends on the position of the appendix. It may be found in either of the lower quadrants or beneath the dia-

phragm (subphrenic abscess). The pus may be discharged through the abdominal wall, the bowel, bladder or vagina, or it may escape into the tissues of the lumbar region or thigh. Appendicitis occasionally excites hepatic abscess, the infection being carried through the portal vein.

Diagnosis.—*Typhoid Fever.*—The gradual onset, characteristic temperature-curve, epistaxis, mental hebetude, diarrhea, splenic enlargement, absence of leukocytosis, and, later, the rash and Widal reaction will indicate typhoid fever.

Renal Colic.—This may be recognized by the absence of fever and of local rigidity, and the presence of hematuria.

Acute Inflammation of the Gall-bladder.—Pain and tenderness in the right hypochondrium, a smooth, mobile tumor, and a history of biliary colic would suggest this condition.

Tubal Disease.—The history and results of pelvic examination will usually prevent an error in diagnosis.

Pneumonia with the pain referred to the abdomen may usually be recognized by the high temperature, frequent respiration, superficial abdominal tenderness, and the presence of a friction-sound or of râles and the affected lung.

Prognosis.—The average mortality is about 14 per cent. With early operation it is less than 1 per cent.

Treatment.—The patient should be kept in bed at absolute rest. All food should be withheld until the acute symptoms have subsided. Constipation is best relieved by enemas of warm water. Locally, cold or heat may be applied according to the sensations of the patient. The use of morphin is generally discredited, since it tends to mask the symptoms.

If the patient is seen within the first forty-eight hours and peritonitis is absent or is limited to the region of the appendix operation should be undertaken at once. After the super-vention of generalized peritonitis it is advisable to delay operation with the hope that the process will result in localized abscess formation, following, in the meantime, Ochsner's treatment, which consists in maintaining the Fowler position,

withholding food, water, and medicines by the mouth, washing out the stomach, applying heat or cold to the abdomen, and giving saline solution freely by the rectum or subcutaneously. Localized abscesses should be opened and drained.

INTESTINAL OBSTRUCTION

(Ileus)

Intestinal obstruction may be either acute or chronic. The chief causes of the *acute form* are: (1) Strangulation; (2) intussusception; (3) volvulus; (4) impaction of foreign bodies or gall-stones; (5) paresis of the intestine; (6) congenital malformation or stricture.

Chronic obstruction may be due to—(1) Impaction of feces; (2) stricture; (3) tumors of the bowel or of adjacent organs.

Symptoms of Acute Obstruction.—These consist of: (1) Sudden abdominal pain—at first paroxysmal, but later continuous; (2) constipation, soon becoming absolute; (3) vomiting, persistent and ultimately of a stercoraceous character; (4) abdominal distention; (5) visible peristaltic waves; (6) collapse, indicated by pinched features, sunken eyes, a cold, clammy surface, and a frequent feeble pulse.

Symptoms of Chronic Obstruction.—The symptoms usually develop gradually. Acute symptoms may appear, however, when the occlusion becomes complete. The chief features are intractable constipation, colicky pains, distention of the abdomen, and gradual failure of health. The stools may be ribbon-shaped or in the form of scybalous masses, and are sometimes coated with mucus and blood. Vomiting is not common.

Diagnosis.—Early vomiting, slight distention, suppression of urine, and rapid collapse point to an obstruction *high in the small intestine*.

Acute Generalized Peritonitis.—The history, early appearance of fever and of diffuse tenderness, signs of effusion, and absence of stercoraceous vomiting will indicate peritonitis.

Strangulation.—This often occurs in external hernia, when it can be recognized by an examination of the inguinal, femoral, and umbilical rings.

Internal strangulation is very common. It may be due to the slipping of a coil of intestine under bands of adhesions, the result of antecedent peritonitis, or under Meckel's diverticulum that is abnormally attached to the abdominal wall, or through a slit in the omentum or mesentery, the foramen of Winslow, or the diaphragm. It usually occurs in young adults; there is often a history of injury or of peritonitis, and the symptoms are very acute.

Intussusception or Invagination.—This is the slipping of a portion of the intestine into the part immediately below it. It occurs especially in children. Its exciting cause is probably irregular peristalsis, whereby one part of the bowel is constricted while the adjoining part is dilated. The usual seat is the ileocecal region.

Multiple invaginations are frequently found postmortem, which have resulted from the irregular peristalsis occurring just before death; they possess no inflammatory characteristics. In invaginations not cadaveric the parts are injected, swollen, and covered with fibrin.

The age of the patient, the sudden abdominal pain, the vomiting, the passage with tenesmus of mucus and bloody feces, and the presence of a sausage-shaped tumor in the region of the descending colon are the diagnostic features. Occasionally the invaginated portion can be felt in the rectum.

Death usually results from gangrene, peritonitis, or collapse. A favorable termination sometimes results from the escape of the incarcerated part, or by a sloughing off of the strangulated portion and adhesion of the serous surfaces.

Volvulus or Twist or Knot of the Bowel.—Volvulus occurs most commonly in middle-aged men. The usual seat is the sigmoid flexure. A relaxed and lengthened mesentery is a predisposing factor. It cannot be recognized with certainty without abdominal section.

Impaction of Foreign Bodies.—Foreign bodies swallowed by accident or design, gall-stones, or enteroliths may cause acute intestinal obstruction. The history may aid in the diagnosis.

Gall-stones ileus is most frequently met with in women after the fiftieth year. The ileocecal region is the usual seat of the obstruction.

Paresis of the Bowel.—This may also result from peritonitis, an abdominal operation, the reduction of a hernia, or traumatism.

Congenital Malformation.—This rare form of obstruction usually consists in an imperforate condition of the anus or rectum. It may be recognized by digital examination.

Impaction of Feces.—This may occur at any age, but it is most often seen in persons past middle life. The usual seat of the impaction is the rectum or colon. The condition may be recognized by the gradual onset of the symptoms, the history of habitual constipation, and by the presence of a fecal mass in the rectum or of an irregular, painless, doughy tumor in the region of the colon.

Stricture and Tumors.—Cicatricial contraction may result from syphilitic, tuberculous, or dysenteric ulceration. The rectum is the part most frequently involved. The most common tumor of the bowel is cancer. It is most often seated in the rectum. The diagnosis may be established by the history of the case, the gradual onset of obstructive symptoms, impairment of health, painful defecation, the size and form of the stools, the presence of blood and pus in the stools, and the results of a physical examination.

Treatment.—*Acute Obstruction.*—Food by the mouth should be withheld. Ice may be given to quench thirst. *Cathartics are contraindicated.* Pain is best relieved by warm applications and the administration of morphin hypodermically. Washing out the stomach three or four times daily is recommended for the persistent vomiting. Distention of the large bowel with warm water or gas should be practised in doubtful cases

and in intussusception. It is best done under anesthesia with the patient in knee-elbow position. After failure with these methods operation should not be delayed; the earlier its performance, the greater the chance of success.

Chronic Obstruction.—The treatment will vary with the cause. Surgical intervention is frequently required.

In fecal impaction injections of warm water, of oil (4 to 6 fluidounces), or of aqueous solutions of ox-gall (2 drams to 1 pint) are efficient. Salines may be administered by the mouth. Massage is sometimes useful. Hard rectal accumulations may have to be removed by the fingers or a suitable scoop.

DISEASES OF THE PANCREAS

ACUTE PANCREATITIS

Varieties.—Hemorrhagic, suppurative, and gangrenous.

Etiology.—Acute pancreatitis may result (1) from cholelithiasis, due to the extension of an infectious inflammation from the biliary tract into the pancreatic duct or to the entrance of bile into the pancreas, as when a gall-stone is so lodged in the diverticulum of Vater that the common bile-duct and the pancreatic duct become a continuous closed channel; (2) from inflammatory affections in adjacent parts—gastro-duodenal catarrh, gastric ulcer, or cancer; (3) from general infections—specific fevers and pyemia; (4) from traumatism. Many of the patients are fat and have used alcohol in excess.

Pathology.—In the hemorrhagic form the organ is irregularly enlarged and the seat of hemorrhagic extravasation. Opaque, white spots of a tallowy consistence are frequently found in the interlobular tissue, omentum, and surrounding parts, and represent areas of *fat necrosis*.

Suppurative pancreatitis may occur as a primary condition or as a sequel of the hemorrhagic form. There may be multiple abscesses or one large collection of pus. More or less extensive areas of necrosis are found. Thrombosis of the portal or splenic veins is frequently encountered. Pancreatic abscesses may become encapsulated or they may rupture into the peritoneum, stomach, or duodenum.

Gangrenous pancreatitis is usually secondary to one of the other varieties.

Symptoms.—Hemorrhagic pancreatitis is marked by sudden intense pain in the upper part of the abdomen often radiating to the back; distention of the epigastrium, with localized tenderness and rigidity; vomiting of bile-stained mucus, or occasionally, of bloody material, and symptoms of profound collapse. Constipation is the rule, but diarrhea is not uncommon. Slight jaundice is often observed. Fatty stools and glycosuria are very rarely present. Death usually occurs in from one to three days, but sometimes the severity of the symptoms diminishes and the disease enters upon a stage of necrosis (gangrene) or of suppuration extending over several weeks or months. This transition is indicated by a tumor mass in the epigastrium, irregular fever, leukocytosis, and progressive weakness and emaciation. Jaundice and chills may also occur.

Occasionally in primary suppurative pancreatitis the onset is gradual, and for many months the only symptoms are abdominal pain and digestive disturbances. In other cases, however, jaundice, fever, chills, diarrhea, emaciation, and a tumor mass in the epigastrium are also present.

Diagnosis.—*Intestinal Obstruction.*—In this condition the onset is usually less severe, fecal vomiting is common, pain and distention are less frequently limited to the epigastrium, and constipation is absolute, not even flatus being passed.

In *perforation of a gastric or duodenal ulcer* the antecedent history usually presents some of the characteristic features of ulcer, and although the patient's face is pallid and anxious, his pulse, at least in the first two or three hours, is very much less altered either in frequency or volume than in acute pancreatitis. In *biliary colic* the symptoms of collapse are less pronounced, the pain, tenderness, and rigidity are chiefly in the region of the gall-bladder, and the extreme restlessness of the patient are in striking contrast with the motionless rigidity observed in acute pancreatitis. In *ruptured extra-uterine pregnancy* the history, the results of vaginal examination, and the rapidly increasing pallor and faintness with sighing inspiration, restlessness, etc., will usually determine the diagnosis.

Prognosis.—This is very unfavorable. The duration varies from a day or two in the hemorrhagic form, to several weeks in the chronic suppurative variety. Recovery may follow operation or rupture of the abscess into the bowel. The disease rarely ends in chronic pancreatitis.

Treatment.—Operation at an early period offers some hope of cure.

CHRONIC PANCREATITIS

(Cirrhosis of the Pancreas)

Etiology.—It may result—(1) From closure of the pancreatic duct by gall-stones impacted in the common bile-duct; (2) from extension of inflammation in gastro-duodenal catarrh or pyloric ulcer; (3) from syphilis or alcoholism; (4) from sclerosis of the pancreatic arteries, and, possibly, (5) from acute pancreatitis.

Pathology.—The chief lesions are an overgrowth of the fibrous tissue and more or less degeneration or atrophy of the cellular elements.

Symptoms.—The symptoms are obscure. Flatulent dyspepsia, paroxysmal epigastric pain, a tendency to diarrhea, slight jaundice, and progressive wasting are the usual features. Fatty stools have been noted in some instances. When the cirrhotic process is diffuse (chronic interacinous pancreatitis) and implicates the islands of Langerhans, the symptoms of *diabetes mellitus* develop, otherwise glycosuria is rare.

Prognosis.—The disease runs a slow course. If glycosuria develops, the outlook is more grave.

Treatment.—The use of fats and starches should be restricted. Carbonated waters are said to increase pancreatic secretion. Pancreatin is recommended. Surgical treatment offers a good chance of recovery in gall-stone cases.

CARCINOMA OF THE PANCREAS

Etiology.—The disease most frequently occurs in males past forty years of age.

Pathology.—Pancreatic cancer generally involves the head of the gland, and is commonly of the scirrhus variety. It may be primary or secondary.

Symptoms.—These include disturbances of digestion, rapid loss of flesh and strength, anemia, severe deep-seated epigastric pain, and the presence of a tumor. The latter is usually found a little above the navel; it is but slightly movable, deep seated, and often pulsatile from its relation to the aorta. The pain often occurs in paroxysms, especially at night, and may be associated with the symptoms of collapse. Progressively increasing jaundice, with enlargement of the gall-bladder, is a frequent symptom, and results from the pressure of the tumor upon the common bile-duct. Pressure on the portal vein may cause ascites. Glycosuria is an occasional symptom. In some cases the stools have contained much free fat and numerous undigested muscle-fibers.

Diagnosis.—*Gastric Cancer.*—In this condition the tumor is more freely movable, is usually associated with dilatation of the stomach and with marked gastric symptoms. Pain is not usually so severe. Jaundice is uncommon.

Gall-stone in Common Duct.—In this condition jaundice is likely to be remittent, febrile paroxysms are often present, there is no epigastric tumor, the gall-bladder is usually small, and emaciation is less rapid.

CYSTS OF THE PANCREAS

Varieties.—(1) Retention cysts from impaction of a calculus, stricture, or tumor; (2) traumatic cysts from hemorrhagic extravasation; (3) proliferation cysts (carcinomatous or adenomatous).

Pathology.—Pancreatic cysts, as a rule, grow forward and upward, and project between the stomach and colon. The contents, which are commonly viscid, may be clear or bloody. Pancreatic ferments are often present.

Symptoms.—These are very variable, the most common being deep-seated epigastric pain, digestive disturbances,

vomiting, and emaciation. Jaundice sometimes occurs from compression of the bile-duct, and less frequently ascites from compression of the portal vein. Free fat and undigested muscular fiber may be found in the stools and sugar in the urine. Physical examination often reveals in the upper part of the abdomen, between the stomach and the colon, a smooth, rounded, fluctuating tumor.

Prognosis and Treatment.—The prognosis is guardedly favorable under operative treatment.

PANCREATIC CALCULI

Pancreatic calculi are probably due to infection of the pancreatic ducts and stagnation of the pancreatic secretion. Their passage through the duct excites *pancreatic colic*, the symptoms of which resemble biliary colic but the pain is more likely to radiate to the left and is usually unattended with jaundice. The coexistence of glycosuria, with fatty stools, and the discovery in the stools of concretions containing chiefly carbonate or phosphate of lime, would confirm the diagnosis.

DISEASES OF THE LIVER

Area of Liver Dulness.—The absolute dulness (part uncovered by lung) extends in the mammary line from the upper border of the sixth rib to the costal margin; in the axillary line, from the seventh rib to the eleventh rib; in the scapular line, from the ninth rib to the eleventh rib; in the median line, the upper border is lost in the cardiac dulness, while the lower border lies midway between the ensiform cartilage and the umbilicus. Slight dulness in the mammary line begins at the fifth rib.

Palpation.—*Palpation* of the liver is practiced to determine position, size, form, and consistence and to detect any tenderness or pulsation.

Conditions in which the liver is palpable:

1. In thin subjects the edge is sometimes palpable under normal conditions.
2. In very young children in whom the liver is always disproportionately large.
3. In depression of the liver, as by a pleural effusion or by a consolidated lung.
4. When the suspensory ligaments are relaxed and the liver "wanders."
5. In enlargement of the organ from any cause.
6. In certain abnormalities of form, as in the "corset liver."

Superficial Irregularities.—Small irregularities may be noted in cancer of the liver, syphilis of the liver, and very rarely in atrophic cirrhosis.

Large prominences are sometimes noted in tumors, abscesses and hydatid cysts.

Consistence.—The liver is firm to the touch in hypertrophic cirrhosis, cancer, congestion, leukemic infiltration, and amyloid disease. In abscess and hydatid disease the resistance is less marked and sometimes fluctuation can be noted.

Tenderness.—The liver is often tender in congestion, abscess, cancer, hypertrophic cirrhosis, and in affections complicated with perihepatitis.

Pulsation may be detected in the venous congestion resulting from tricuspid regurgitation, in abdominal aneurysm, and in tumors of the left lobe resting on the aorta.

Percussion.—Percussion determines size and resistance.

The liver is uniformly enlarged in: (1) Congestion, active and passive. (2) Fatty infiltration. (3) Amyloid infiltration. (4) Cirrhosis, especially hypertrophic. (5) Leukemic infiltration. (6) Infiltrating carcinoma. (7) Suppurative cholangitis.

Irregular enlargements of the liver are often noted in: (1) Nodular cancer. (2) Abscess. (3) Hydatid disease. (4) Syphilis.

The liver is diminished in size in: (1) Atrophic cirrhosis,

late stage. (2) Fatty degeneration. (3) Acute yellow atrophy. (4) Senile atrophy. The area of hepatic dulness may be diminished from certain extrinsic causes, namely, pulmonary emphysema, excessive tympanites, and perforation of the stomach or bowel.

JAUNDICE OR ICTERUS

Definition.—Pigmentation of the tissues and excretions with bile-pigments.

Varieties.—(1) Obstructive jaundice. (2) Hematogenous jaundice.

Etiology of Obstructive Jaundice.—Obstruction to the outflow of bile leads to its accumulation in the biliary passages and reabsorption into the blood.

Obstruction may be due to the following causes:

1. Stricture of the bile-duct, congenital or acquired.
2. Catarrh of the bile-ducts or of the duodenal mucous membrane around the orifice of the ductus choledochus.
3. Foreign bodies in the ducts, as gall-stones or parasites.
4. Tumors of the liver or of adjacent viscera compressing the ducts.
5. Compression of the ducts by enlarged lymph glands, inflammatory adhesions, or fecal accumulations.
6. Kinking of the ducts the consequence of gastropptosis, nephropptosis, abdominal tumors, the pregnant uterus, etc.
7. Spasm of the bile-ducts. This has been advanced as the cause of the jaundice that rarely follows emotional excitement.

Symptoms.—The skin, mucous membranes, and secretions are stained yellow. The discoloration is usually first noticed in the conjunctivæ. The stools are pale, fetid, and fatty; the urine is dark, and in long-standing cases may contain hyaline casts. The pulse rate is often reduced, and the temperature may be slightly subnormal. There is more

or less mental depression, and in chronic cases delirium, convulsions, and coma occasionally develop. Itching of the skin is often noted, and urticaria is a common complication. In grave cases subcutaneous ecchymoses may appear. The clotting time of the blood is increased.

Diagnosis.—Other discolorations, as the bronze hue of Addison's disease, the sallow complexion of advanced cachexia, the pigmentation of the skin in hemochromatosis and the green tint of chlorosis, may resemble jaundice, but in these cases the conjunctiva remains white and the urine lacks bile.

Etiology of Hematogenous Jaundice.—This form of jaundice occurs (1) in certain intoxications as in poisoning by toluyliendiamin, phosphorus or snake-venom; it may be observed (2) in various infections, such as yellow fever, relapsing fever, septicopyemia, etc.; it occurs (3) in acute yellow atrophy of the liver; (4) it occurs in hemolytic jaundice with splenomegaly, congenital and acquired; (5) it is occasionally seen in pernicious anemia and paroxysmal hemoglobinuria.

In hematogenous jaundice there is also obstruction, but it is situated in the minute bile-ducts instead of in the larger ones. The chief cause of the obstruction is probably catarrhal inflammation (cholangitis), excited by the poison circulating in the blood, although extensive destruction of the red blood-cells, which leads to the secretion of a thick, viscid bile, rich in pigments (polychromia), is doubtless a contributing factor in many cases.

Symptoms.—These are much the same as in obstructive jaundice, but the staining of the skin is usually not so intense, and the stools still contain bile. Grave constitutional symptoms (delirium, stupor, multiple hemorrhages, etc.) are more frequent, and other evidences of an underlying infection or intoxication are usually present.

ICTERUS NEONATORUM

Physiologic icterus in the new-born is slight. According to Quincke, it is probably due to the passage of a part of the

portal blood rich in bile-pigments directly into the vena cava by way of the ductus venosus, which remains patent for several days after birth.

Pathologic icterus in the new-born is marked, and commonly proves fatal. It results from congenital stricture of the bile-ducts, syphilis of the liver, septic infection through the umbilical vein, or epidemic infection (Winckel's disease).

CHOLEMIA

The term cholemia has been used to designate the grave intoxication which sometimes develops in severe forms of jaundice and in the later stages of liver disease even in the absence of jaundice. The condition is characterized by headache, delirium, stupor, coma, convulsions, and not rarely, by hemorrhages into the skin and from the mucous membranes. These symptoms are probably due to the retention in the blood of poisonous compounds which are normally rendered non-toxic by the liver, although an underlying or concomitant infection may be a factor in some cases.

ACHOLURIC JAUNDICE

A yellowish discoloration of the skin and conjunctivæ is sometimes seen in cases of pronounced urobilinuria in which the urine is free from bile. This discoloration is not due to urobilin pigmentation, however, but is a true jaundice (*acholuric jaundice*), bile pigment being invariably present in the blood but in quantities too small to be excreted in the urine. Jaundice of this type is the most conspicuous symptom in *chronic family jaundice*, a comparatively rare disease, which tends to affect several members of a family and is usually congenital (Minkowski-Chauffard type), although it may attack but one of a family and may appear first in adolescence (Hayem-Widal type). The disease is characterized by persistent jaundice, enlargement of the spleen, moderate or pronounced anemia, and attacks of acute indigestion or of abdominal pain, due in some instances to complication with

gall-stones. The stools are well colored and the urine contains urobilin but no bile. The liver is not at all or only slightly enlarged and signs of constitutional disturbances are often lacking. The symptoms are probably due to increased fragility of the erythrocytes. Other conditions that must be ruled out in making the diagnosis are congenital stenosis of the bile-ducts, the infectious forms of icterus, syphilitic hepatitis and splenic anemia. So far as life is concerned the prognosis is good. In many instances splenectomy has effected a cure. X-ray has sometimes been of service.

CATARRHAL CHOLANGITIS

(Catarrhal Jaundice; Catarrh of the Bile-ducts)

Etiology.—(1) The primary form is usually met with in children or young adults as a sequel of a gastro-intestinal catarrh, which has been excited by dietetic indiscretions or exposure to cold. (2) The disease is an occasional accompaniment of infectious diseases, as pneumonia and typhoid fever. (3) It is often associated with cholelithiasis or organic disease of the liver. (4) It sometimes occurs in epidemic form.

Pathology.—The large ducts are particularly affected; the mucous membrane is swollen and covered with tenacious mucus. When the gall-bladder is compressed, bile is ejected through the duodenal orifice with less ease than is natural.

Symptoms of Acute Catarrhal Cholangitis.—(1) Symptoms of gastro-duodenal catarrh usually usher in the disease. These are: Coated tongue, anorexia, feter of breath, epigastric discomfort, vomiting, and perhaps diarrhea. (2) Obstructive jaundice, indicated by yellow skin and conjunctivæ, light stools, and dark urine, is a constant symptom. (3) In some cases there is slight fever with swelling and tenderness of the liver.

Diagnosis.—This is based upon the acute course, the mild character of the symptoms, the history of preceding gastric catarrh, and the youth of the patient.

Prognosis.—This is, as a rule, favorable. The average duration of the disease is from two to six weeks.

Treatment.—The diet should be simple and digestible. Fatty and saccharine foods should be avoided. Milk, broths, eggs, lean meats, oysters, and well-cooked cereals are admissible. Sodium phosphate (1 dram three times a day), silver nitrate ($\frac{1}{4}$ grain three times a day), and ammonium chlorid (5 to 10 grains three times a day) are of value in relieving the primary gastro-duodenal catarrh. In obstinate cases nitrohydrochloric acid may prove beneficial. Daily irrigation of the colon with from 1 to 2 quarts of cold water is sometimes of service. Free water-drinking between meals is to be recommended. Alkaline mineral waters (Vichy, Vals, Hathorn) often act well.

Chronic catarrhal jaundice may follow repeated acute attacks, but in the large majority of cases it is a sequel of stenosis of the common bile-duct from gall-stones, stricture, or pressure from without. A constant symptom is chronic jaundice. Jaundice developing gradually without pain and increasing steadily from week to week, while the gall-bladder increases in size, is suggestive of *compression of the common duct by a tumor or cicatricial tissue*; persistent jaundice of varying intensity, preceded by colicky pains, and accompanied by ague-like paroxysms of a fever chill and sweat, is suggestive of *obstruction of the common duct by gall-stone*.

SUPPURATIVE CHOLANGITIS

Etiology.—Suppurative inflammation of the biliary ducts is usually a sequel of gall-stones or of obstruction of the ducts by tumor. Occasionally it follows a general infection, such as typhoid fever or pneumonia. The chief provoking organisms are the pyogenic cocci, the colon bacillus, and the typhoid bacillus.

Symptoms.—These comprise the usual phenomena of sepsis (fever, chills, sweats, and leukocytosis), with jaundice,

local discomfort or actual pain, enlargement of the liver, perhaps also of the spleen, and emaciation. The gall-bladder is generally distended from concomitant cholecystitis.

The disease is a grave one, and surgical intervention offers the only chance of cure.

ACUTE CHOLECYSTITIS

Definition.—Acute inflammation of the gall-bladder.

Etiology.—The disease is always infectious, the organisms most commonly present being the colon bacillus, typhoid bacillus, pneumococcus, staphylococcus, and streptococcus. Injury to the mucosa by gall-stones is an important predisposing factor. It is not an uncommon complication of infectious fevers, especially typhoid fever.

Pathology.—The inflammation may be catarrhal or suppurative. Suppurative cholecystitis (*empyema of the gall-bladder*) is usually associated with purulent inflammation of the bile-ducts, and, unless promptly relieved by operation, proceeds to ulceration or gangrene and general peritonitis.

Symptoms.—In catarrhal cases the symptoms are slight fever, pain in the hepatic region, tenderness and enlargement of the gall-bladder, and, occasionally, jaundice. In the suppurative form there are severe paroxysmal pain, vomiting, a septic type of fever, leukocytosis, enlargement and tenderness of the gall-bladder, and, in some cases, jaundice.

Diagnosis.—It must be distinguished from *appendicitis*, *subphrenic abscess*, and *acute pancreatitis*. The discriminating features are the history of previous cholelithiasis, typhoid fever, or pneumonia, and the locality of the pain, tenderness, and swelling.

Prognosis.—Catarrhal cholecystitis usually subsides in from one to three weeks, but gall-stones and adhesions between the gall-bladder and adjacent organs are common sequels. In suppurative cases operation affords a reasonable hope of benefit.

CHOLELITHIASIS

(Gall-stones; Biliary Calculi)

Etiology.—Gall-stones are three or four times more common in women than in men. They occur most frequently after middle life, and are rarely seen before twenty-five. Sedentary habits, high living, tight lacing, obstruction of the ducts, and other factors that favor stagnation and inspissation of the bile predispose to their formation. Their occurrence after typhoid fever and other infections is especially common. The immediate cause of cholelithiasis seems to be a mild catarrhal inflammation of the biliary tract set up chiefly by typhoid or colon bacilli. This catarrh leads to the increased formation of cholesterin and to the precipitation of bilirubin-calcium. The latter, according to Naunyn, is the cementing substance which binds together the cholesterin, desquamated epithelium, agglutinated bacilli, etc.

Pathology.—Gall-stones may be found in the ducts, but in the large majority of cases they originate in the gall-bladder. There may be one or several hundred. When multiple, they are found with facets, from attrition. The size varies from that of a grain of sand to that of a large walnut. The color varies from light yellow to dark green. The chief constituent is cholesterin, but bile-pigments and lime salts may also enter into their composition. On section, they usually present a concentric arrangement.

Events.—(1) Gall-stones may remain quiescent or give rise to no symptoms other than those of continued or recurring indigestion, with ill-defined pains in the upper part of the abdomen. (2) In consequence of violent expulsive efforts, excited by irritation of the gall-bladder, they may be extruded into the bowel, intense pain (*biliary colic*) marking their passage through the ducts. (3) Instead of making a complete exit, they may slip back into the gall-bladder or they may become impacted in the cystic duct, or, more often, in the lower part of the common duct. (4) They may perforate into

the duodenum, peritoneum, lung, stomach, or kidney, or externally. Perforation may be followed by stricture of the ducts or by fistulous communications between the ducts and adjacent viscera. Perforation into the duodenum is not a rare cause of intestinal obstruction. (6) Although gall-stones are the result of a mild catarrh of the gall-bladder, their presence favors fresh infection and, therefore, the occurrence of active, even suppurative, cholecystitis or cholangitis. From the gall-bladder the inflammation may spread to the peritoneum, and if acute, result in a circumscribed abscess, or if chronic, lead to the formation of adhesions, which in turn may occasion digestive disturbances and colicky pains or even give rise to gastrectasis or intestinal obstruction. (7) Pancreatitis, acute or chronic, sometimes supervenes from extension of infection through the lymphatics or the biliary and pancreatic ducts. (8) The prolonged irritation excited by calculi may ultimately lead to carcinoma of the biliary passages.

Symptoms of Biliary Colic.—(1) The attacks begin abruptly with intense pain radiating from the hypochondriac region to the right shoulder. There are often tenderness and rigidity over the gall-bladder. Chill and fever (102° – 103° F.) often mark the onset. The symptoms of intense pain are obvious—anxious face, cold sweat, feeble pulse, and vomiting. Jaundice may follow from obstruction, but it is often absent. If the stone escapes, it may subsequently be found in the stools. The attack may last from a few hours to several days.

Diagnosis.—*Renal Colic.*—In this affection the pain radiates from the lumbar region along the ureter into the bladder and genitals. Frequent micturition is a common symptom. There is no jaundice. Blood or the stone may be found in the urine.

Intestinal colic produces pain that radiates around the umbilicus. There are flatulence and borborygmi. Jaundice is absent.

Gastralgia.—Pain is over the whole stomach, does not radiate to the shoulder, and is relieved by pressure. There is no jaundice.

Gastric Ulcer.—Pain is closely related to eating. There are localized tenderness in the epigastrium, hyperacidity, and frequently hematemesis.

Symptoms of Obstruction of the Cystic Duct.—Obstruction of the cystic duct may be followed by cholecystitis (catarrhal, suppurative, or gangrenous), by atrophy of the gall-bladder, or by dropsy of the gall-bladder (*hydrops vesicæ felleæ*). In the last condition the gall-bladder can often be felt as a pear-shaped, elastic, movable tumor, projecting from the lower margin of the liver. Jaundice is not present, and subjective symptoms are slight.

Symptoms of Obstruction of the Common Duct.—In typical cases the symptoms are—(1) Chronic jaundice showing marked variations in intensity; (2) pain, also subject to distinct exacerbations; (3) recurrent attacks of intermittent fever, with chills and sweats (Charcot's hepatic fever). The liver is not enlarged; the gall-bladder is usually not distended, but often atrophied from antecedent attacks of cholecystitis (Courvoisier's law). Obstruction of the common duct may persist for months or years. It not infrequently leads to suppurative angiocholitis, to obstructive biliary cirrhosis, or to acute or chronic pancreatitis.

Diagnosis.—*Obstruction of the Common Duct from Without (Cancer).*—The jaundice increases steadily and is without remission, the gall-bladder is enlarged, and characteristic colic and hepatic fever are wanting.

Prognosis.—In the absence of complications the prognosis of cholelithiasis is good. It must be borne in mind, however, that grave complications (suppurative cholecystitis or angiocholitis, perforation, hemorrhagic pancreatitis) may arise most unexpectedly.

Treatment.—Efforts must be directed to keeping the stones quiescent by preventing irritation or catarrh of the gall-bladder. The food should be plain and readily digestible. Saccharine matters, fat meats, and highly seasoned dishes should be avoided. Water-drinking between meals should

be encouraged. Regular exercise in the open air, provided the symptoms are latent, is extremely beneficial.

Digestive disturbances should receive appropriate treatment. Among drugs, alkalis and alkaline mineral waters are undoubtedly efficacious. Sodium bicarbonate or sodium phosphate may be taken well diluted in the morning an hour before breakfast and also between meals. If there is decided constipation, a small quantity of Rochelle salt or sodium sulphate may be added to each potation. The natural mineral waters, notably those of Carlsbad and Vichy, have acquired a high reputation. When there is a tendency to so-called bilious attacks, an occasional course of calomel in fractional doses will be found of benefit.

Surgical intervention is called for: (1) When, despite medical treatment, attacks of colic occur so frequently and are of such severity as to cause disability or make the addiction to morphin a likelihood; (2) in persistent obstruction of the common duct; (3) in hydrops of the gall-bladder due to impaction or stricture of the cystic duct; and (4) in suppurative inflammation of the gall-bladder or gall-ducts.

Hepatic Colic.—Morphin ($\frac{1}{4}$ grain) and atropin ($\frac{1}{120}$ grain) should be given hypodermically. Agonizing pain often yields very promptly to a few whiffs of chloroform. In the mild but rather persistent attacks a few doses of antipyrin in hot water may suffice. The external application of heat (poultice or hot bath) is useful.

When vomiting is urgent, carbonated water or champagne may be given. In threatened collapse diffusible stimulants are needed.

Obstruction of the Common Duct.—The measures best suited for promoting the advance of the stone into the bowel are rest, regulation of diet, the free use of alkaline mineral waters, the occasional exhibition of saline laxatives, and the application of heat to the hypochondriac region. Olive oil has been recommended as a special remedy, but it is of doubtful efficacy. As the sequelæ of impaction of the common duct are so grave,

surgical aid should be invoked if the obstruction is not removed under medical treatment within a period of three or four weeks.

HYPEREMIA OF THE LIVER

Varieties.—(1) Active hyperemia. (2) Passive hyperemia.

Etiology.—*Active hyperemia* is commonly due to dietetic indiscretions. It may result from overindulgence in alcohol. It is often present in the infectious fevers. It appears to arise idiopathically in hot climates.

Passive hyperemia results from diseases that obstruct the venous circulation, as chronic heart and lung disease.

Pathology.—The liver is enlarged and filled with blood. In the *passive* variety, the center of the lobule, the area of the hepatic vein, is deeply pigmented, while the periphery, the area of the portal vein, is pale. This mottled appearance has given rise to the term “nutmeg liver.” In persistent cases pigmentation, atrophy of the liver-cells, and overgrowth of the connective tissue result—a condition termed “cyanotic induration.”

Symptoms.—*Active Hyperemia.*—The liver is enlarged and somewhat tender. There is a sense of fulness or even actual pain in the hepatic region. There may be slight jaundice. Digestive disturbances—anorexia, nausea, flatulence, headache, and epigastric tenderness usually coexist.

In the *passive variety* the symptoms are much the same as in the active form, but in addition there is frequently ascites. The liver is often quite large, and in extreme cases, such as follow tricuspid regurgitation, it may pulsate.

Prognosis.—In simple active congestion the prognosis is good. In passive congestion the prognosis depends upon the cause.

Treatment.—Active hyperemia from dietetic errors usually yields promptly to restriction of the diet and the administration of a mercurial purge, followed by a saline—Rochelle salt, Seidlitz powder, or sodium phosphate.

In passive congestion treatment must be directed to the primary disease. In mild cases alkaline mineral waters (Carlsbad, Congress, and Friederichshall) do well. A mercurial laxative may be used from time to time. In severe cases the most effective measures are absolute rest, a milk diet, saline purges, and wet-cupping over the liver.

CIRRHOSIS OF THE LIVER

(Chronic Interstitial Hepatitis)

Definition.—A chronic disease of the liver characterized by a hyperplasia of the connective tissue and more or less extensive retrograde changes in the liver-cells.

Varieties.—The most important varieties are *atrophic* or *portal cirrhosis*, *hypertrophic biliary cirrhosis* (*Hanot type*), *obstructive biliary cirrhosis*, *syphilitic cirrhosis*, and *capsular cirrhosis*.

ATROPHIC CIRRHOSIS

(Laennec's Cirrhosis; Alcoholic Cirrhosis; Gin-drinker's Liver)

Etiology.—It occurs most commonly in males of middle age. The chief cause is the continued use of alcohol, especially in the form of raw spirits. It has been suggested that poisonous food products, bacterial toxins or even micro-organisms themselves absorbed from the intestines may also produce this disease. It is possible that some cases owe their origin to the specific fevers.

Pathology.—The liver may be very small, but in many cases it is considerably larger than normal. It is dense and inelastic. The surface is often studded with numerous roundish elevations ("hobnails"). The cut surface, which is usually yellowish in color, displays a network of fine and coarse pearly bands of connective tissue.

Microscopic examination reveals an increased amount of connective tissue, commonly of a fibrous character and chiefly interlobular or periportal in distribution. The paren-

chymatous cells, especially those about the periphery of the lobules, show various degenerative changes, and in advanced cases marked atrophy. On the contrary, evidences of proliferation of the liver-cells, as well as the bile-ducts, are usually found, even in late stages of the disease. It is probable that destruction of certain liver cells through the activity of the poison is the primary change, and that the fibrosis and proliferation of the liver cells are secondary features.

Symptoms.—Obstruction of the portal circulation first causes *congestion and catarrh of the stomach*, hence the initial symptoms are anorexia, fetor of the breath, fulness and distress after eating, eructations, nausea, vomiting of mucus, flatulence, and constipation. For months and even years these phenomena may be the only evidence of the disease. As the pressure in the portal system increases, the collateral vessels enlarge, and as a result the *superficial abdominal veins become prominent* and *hemorrhoids* develop. Engorgement of the portal system also leads to *ascites* and swelling of the feet, to *enlargement of the spleen*, and, not infrequently, to copious *hemorrhage* from the stomach or bowel.

The size of the liver varies; it may be increased or diminished. There is a gradual loss of flesh and strength. The skin is muddy in appearance, but conspicuous jaundice is not common and occurs only as a complication. Nervous symptoms—delirium, stupor, convulsions, and coma—occasionally appear toward the end of the disease. They are probably due to the retention of poisons that the liver is unable to convert or to eliminate.

The majority of cases terminate fatally in from three to five years, or in from one to two years after the compensatory circulation fails. Death results from exhaustion, hemorrhage, pulmonary edema, intercurrent disease, or toxemia.

Complications.—The kidneys, heart, and blood-vessels are often coincidentally involved in the cirrhotic process. Tuberculosis, especially of the peritoneum, is a common complication.

Diagnosis.—In the early stage the diagnosis can only be suspected. In the drunkard, chronic gastric catarrh with enlargement of the liver would strongly indicate the disease.

Thrombosis of the portal vein produces a similar clinical picture, but the symptoms usually develop much more rapidly.

Chronic Diffuse Peritonitis with Effusion.—This is usually tuberculous or cancerous. The history, abdominal tenderness, the detection of localized masses or ill-defined indurations, the presence of other foci of disease, the high specific gravity (above 1014) of the ascitic fluid, and the absence of symptoms indicating portal obstruction will generally suggest chronic peritonitis.

Splenic Anemia.—This disease is chiefly distinguished by the absence of a history of alcoholic excess, the early appearance and marked degree of the splenic enlargement, and the very pronounced anemia.

Chronic Perihepatitis.—In this disease the etiologic factors of cirrhosis of the liver are absent, the course is slow, the ascites often remains stationary for long periods and returns again and again after tapping, and signs of marked portal obstruction (abdominal varices and hemorrhages) are lacking.

Prognosis.—The outlook for permanent relief is bad.

Treatment.—Alcohol must be interdicted. A diet of bland, readily digested food is indicated. The gastric catarrh should receive appropriate treatment. Lavage of the stomach is contraindicated on account of the presence of esophageal varicosities. Potassium iodid is of service in syphilitic cases, but not otherwise. Ammonium chlorid (10 grains three times a day) is sometimes useful. Portal congestion is best relieved by the administration of salines (sodium phosphate or Rochelle salt) in hot water one-half hour before breakfast.

Ascites can sometimes be removed by the administration of cathartics and diuretics. A concentrated solution of Epsom salts ($\frac{1}{2}$ to 1 ounce), taken in the morning before breakfast, is usually the most efficient purgative. Occasionally it may be desirable to substitute compound jalap powder or elaterium.

The diuretics of approved value are potassium acetate or bitartrate, theobromin, digitalis, and squill.

Guy's or Baillie's pill has a well-deserved reputation:

℞. Massæ hydrargyri
Pulveris digitalis
Pulveris scillæ..... āā gr. xx.—M.
Fiant pilulæ No. xx.
SIG.—One pill thrice daily.

When the ascites is large and does not yield readily to drugs, paracentesis should be practiced (see p. 121).

Surgical Treatment.—Talma's operation (suture of the omentum to the margin of the abdominal incision and irritation of the peritoneal surfaces of the liver) or one of its modifications, has proved of some benefit in a limited number of cases of liver cirrhosis with ascites. The object of the operation is to establish a compensatory circulation by making accidental adhesions and thus increasing the anastomoses between the vessels of the portal system and those of the systemic circulation. The operation is contraindicated if cardiac or renal disease coexists.

HYPERTROPHIC BILIARY CIRRHOSIS

(Hypertrophic Cirrhosis of Hanot)

Etiology.—The causes of hypertrophic biliary cirrhosis are obscure. Alcoholism does not appear to be a factor. An infective origin has been suggested. The disease is seen chiefly in men between twenty and thirty-five years of age.

Pathology.—The liver is greatly enlarged throughout the entire course of the disease. The organ is of a yellowish or greenish color, and its surface is smooth or finely granular. The spleen is enlarged.

Microscopic examination of the liver reveals an overgrowth of connective tissue, but this is delicate and fibrillary, and shows a marked tendency to enter the lobules and form a fine network between the cells (intralobular or monolobular cir-

rhosis). The small bile-ducts show proliferation of their epithelium and often contain plugs of inspissated bile. Many so-called new bile-ducts are seen. The liver cells often show comparatively little change. The chief features distinguishing it from atrophic or portal cirrhosis are the *intralobular* distribution of the fibrosis, the inflammatory changes in the biliary capillaries (radicular cholangitis) and the inconspicuous damage to the liver cells.

Symptoms.—The liver is uniformly enlarged, usually to a marked degree, throughout the course of the disease. Not rarely it is slightly tender. The spleen is also enlarged. Jaundice, varying in degree from time to time, is a conspicuous feature. Periodic attacks (crises) of pain in the hepatic region, attended by fever, leukocytosis, increase of jaundice, and, perhaps, vomiting, are of common occurrence. Toward the end of the disease hemorrhages into the skin and from the mucous membranes and symptoms of hepatic intoxication, such as delirium and stupor, often develop. Ascites, profuse hematemesis and enlargement of the superficial abdominal veins are rarely observed. The prognosis is unfavorable, but the course of the disease is long—often from five to ten years.

Treatment.—The treatment is in the main that suggested for hyperemia of the liver (see p. 103). In a number of instances the gall-bladder has been drained with good results.

OTHER FORMS OF CIRRHOSIS OF THE LIVER

Syphilitic Cirrhosis of the Liver.—In the diffuse form the appearance of the liver is similar to that of alcoholic cirrhosis. In the gummatous form, however, the organ is enlarged and often coarsely lobulated from fibrous transformation of the gummata. Other manifestations of syphilis, a positive Wassermann reaction, an enlargement of the liver that is grossly nodular, and the presence of unexplained fever should suggest the application of the therapeutic test.

Capsular Cirrhosis (Chronic Perihepatitis, Zuckergussleber). This form is characterized by enormous thickening of the

capsule of the liver (sugar-iced liver). The symptoms closely resemble those of atrophic cirrhosis, but the course is extremely slow, the ascites remaining stationary for long periods and returning again and again after tapping. In many cases chronic capsulitis of the spleen, chronic pleurisy, and chronic obliterative pericarditis are also present, constituting the disorder known as multiple serositis, Pick's disease or Concato's disease.

Obstructive Biliary Cirrhosis.—This form closely resembles Hanot's cirrhosis, but there is a history of cholelithiasis or chronic cholecystitis, the jaundice is more intense, the liver is usually only moderately enlarged, and splenomegaly is often absent.

ABSCESS OF THE LIVER

(Acute Suppurative Hepatitis)

Etiology.—Abscess of the liver is always due to the action of micro-organisms—*Entamoeba histolytica*, streptococcus, staphylococcus, colon bacillus. They may enter the liver directly from an adjacent organ or through the portal vein, hepatic artery, bile-ducts, or lymphatics.

(1) Amebic dysentery is a very common cause, the amebæ entering the liver through the portal vein. Occasionally amebic abscesses occur without any evidence of dysentery. (2) Septic emboli from gastric ulcers, duodenal ulcers, purulent appendicitis, etc., may also lodge in the branches of the portal vein and thus excite a suppurative inflammation (suppurative pylephlebitis). (3) Pyogenic organisms may enter the hepatic artery in ulcerative endocarditis, abscess of the lungs, and general pyemia. (4) Suppuration by way of the bile-ducts sometimes occurs in angiocholitis secondary to gall-stones. (5) Traumatism may be a causal factor. (6) Occasionally gastric ulcer, ulcerative cholecystitis, perinephric abscess, or empyema invades the liver and sets up suppuration inside of it. (7) Finally, suppuration may result from the secondary infection of an echinococcus cyst.

Pathology.—The abscesses following amebic dysentery (“tropical abscess”) and traumatism are generally solitary, and usually occupy the right lobe. Metastatic abscesses are multiple.

Events.—Hepatic abscess may kill by septic poisoning or by perforation into the lung, peritoneum, stomach, pleura, pericardium, or vena cava. Recovery may follow operation or spontaneous rupture into the bronchi, into the stomach or bowel, or externally.

Symptoms.—*Local Symptoms.*—The liver is enlarged and tender. The enlargement is more often upward than downward. Circumscribed bulging beneath the costal arch is sometimes noted. Fluctuation is occasionally detected. There is usually severe pain in the liver region and right shoulder. Exploratory puncture may reveal pus. Slight jaundice may develop, but it is often absent.

Constitutional Symptoms.—These result from sepsis, and include fever of a remittent or irregular type, chills, profuse sweating, marked anemia, and leukocytosis.

Rupture into the lung is characterized by severe cough, weak breath sounds at the base of the right lung, and the expectoration of large amounts of pus, often mixed with blood. In the case of amebic abscess the sputum may have a chocolate color and may contain amebæ.

Diagnosis.—*Hydatid cysts* develop slowly, are not painful, are not associated with septic phenomena, and yield clear fluid on aspiration.

Cancer of the Liver.—The history, marked cachexia, involvement of other organs, presence of jaundice, detection of hard nodules on the surface of the liver, and the absence of septic phenomena will suggest cancer.

Intermittent Fever Due to Impacted Gall-stones.—In this condition the pain, fever, and sweating are often periodic; the health in the intervals may be well preserved; the jaundice increases at each paroxysm; the symptoms may persist for several years.

Prognosis.—Embolic abscesses are invariably fatal. Traumatic and amebic abscesses may terminate favorably upon spontaneous or induced evacuation.

Treatment.—In multiple abscesses treatment is palliative. Large solitary abscesses should be opened and drained.

CARCINOMA OF THE LIVER

Etiology.—Cancer of the liver is more common in men than in women. It is infrequent before the age of forty. Heredity, traumatism, and chronic irritation from gall-stones are predisposing factors.

Pathology.—Primary cancer of the liver is rare; secondary cancer is common. The primary form may appear as a single large nodule (*massive cancer*) or as a widespread infiltrating growth (*nodular cancer*). The latter form is sometimes associated with cirrhosis of the liver (*cirrhotic cancer*). The secondary variety is usually due to the lodgement in the portal capillaries of cancerous emboli derived from a primary growth in one of the adjacent organs, especially the stomach. The liver is much enlarged, and studded with numerous grayish-white nodes, some of which project from the surface. The superficial nodes are often depressed at the center.

Symptoms.—(1) The liver is enlarged and painful, and often presents one or more smooth, hard nodules. The latter may show a central depression. (2) Cachexia is pronounced and develops rapidly. (3) Jaundice is common, but it is rarely intense. (4) Digestive disturbances are a prominent feature, and often precede the hepatic symptoms. Ascites sometimes results from portal obstruction. Toward the end, slight fever, delirium, stupor, and coma may develop (hepatic intoxication).

Diagnosis.—*Hypertrophic biliary cirrhosis* may be distinguished by the smooth, uniform enlargement of the liver, the enlargement of the spleen, the persistence of icterus without loss of color in the stools, the absence of marked cachexia,

the age of the patient (between twenty and forty), and the slow course.

Abscess.—This may be distinguished by the history, the septic fever, and the results of exploratory puncture.

Syphilis of the Liver.—Other manifestations of syphilis, a positive Wassermann reaction, and the therapeutic test are the important aids in diagnosis.

Prognosis and Treatment.—The outlook is hopeless. The duration is from a few months to a year. Treatment can only be palliative.

HYDATID CYST OF THE LIVER

(*Echinococcus of the Liver*)

Etiology and Pathology.—Hydatid cysts are formed by the embryos of the *Tænia echinococcus*, a small tapeworm inhabiting the intestines of the dog. The disease is common in Iceland, Australia, and some parts of Europe, but is rare in America.

The eggs of the worm are accidentally ingested by man, and embryos are liberated in the stomach, whence they may migrate to any organ; the liver, however, is most commonly affected through the portal vein. The fixed embryo soon develops into a cyst that is composed of an external laminated layer and an internal breeding layer. A connective-tissue layer is formed on the outside from irritation.

The cyst contains a clear, non-albuminous fluid of low specific gravity (1002 to 1010) and rich in sodium chlorid.

Scolices or larvæ develop from the breeding layer; they are provided with four suckers and a circle of hooklets, and produce daughter-cysts within the parent-cyst. When ingested by the dog, the larvæ develop into mature tapeworms.

Symptoms.—Small cysts excite no symptoms. Large cysts produce an irregular enlargement of the liver, with a sensation of weight or fulness in the hypochondriac region. If the cyst is superficial, an elastic, fluctuating tumor may be detected on palpation. On percussion a peculiar vibratory sensation

(hydatid thrill) may be imparted to the hand. Aspiration yields a clear fluid containing the characteristic hooklets. Fever, pain, and jaundice are usually absent. An increase of the eosinophiles of the blood (5 to 20 per cent.) is the rule.

Events.—(1) The cyst may reach a certain size and then become quiescent. (2) Trifling injury may convert it into abscess. (3) Rupture of the cyst into adjacent organs may terminate in death or in recovery.

Diagnosis.—The diagnostic features are a smooth, tense, elastic tumor of the liver, of slow growth, without pain, fever, or pronounced disturbance of the general health, and yielding, upon exploratory puncture, a clear fluid containing hooklets. Eosinophilia is also of diagnostic aid.

Prognosis.—In uncomplicated cases the prognosis is guardedly favorable.

Treatment.—Aspiration under antiseptic precautions is sometimes followed by permanent recovery. Surgeons of the largest experience, however, prefer free incision and evacuation of the cysts. Purulent cysts should be treated as abscesses.

AMYLOID LIVER

(Waxy Liver; Lardaceous Liver)

Definition.—An enlargement of the liver due to the deposition of a peculiar albuminoid substance.

Etiology.—The chief cause is prolonged suppuration, especially that resulting from tuberculosis or syphilis and involving the bones. Less frequently it occurs in malarial cachexia and leukemia.

Pathology.—The liver is uniformly enlarged, hard, and smooth. The margins are blunt. On section, the surface presents a translucent, wax-like appearance, and is colored mahogany brown with Lugol's solution. The degenerative process begins in the walls of the blood-vessels and spreads to the connective tissue.

Symptoms.—The liver is uniformly enlarged, smooth, firm, and painless, and presents a rounded edge. The spleen and

kidneys almost always share in the degeneration, and, in consequence, the spleen is enlarged and hard and the urine contains albumin and tube-casts. Anemia and emaciation are often pronounced. Jaundice and ascites are uncommon.

Diagnosis.—This is based upon the history, the uniform enlargement of the liver, the absence of pain, of jaundice, and of ascites, and the involvement of other organs. In *leukemia* the liver and spleen are often uniformly enlarged, but an examination of the blood will prevent an error in diagnosis.

Prognosis and Treatment.—The prognosis depends somewhat upon the curability of the primary disease. The outlook, however, is always grave. The treatment must be directed to the causal disease.

ACUTE YELLOW ATROPHY OF THE LIVER

(Acute Parenchymatous Hepatitis; Icterus Gravis)

Definition.—A very rare and grave disease characterized anatomically by a rapid destruction of the liver tissue, and manifested clinically by jaundice, hemorrhages, a reduction in the size of the liver, and marked cerebral phenomena.

Etiology.—The disease occurs more frequently in women than in men. It is usually seen between the ages of twenty and thirty. Pregnancy is the most important predisposing factor. Alcoholic excesses, syphilis, and emotional excitement are also thought to have some etiologic significance. Acute yellow atrophy of the liver appears to be closely related to severe cases of epidemic catarrhal jaundice and to cases in which acute degenerative changes are engrafted on some pre-existing disease of the liver, such as cirrhosis. The rapid course, widespread lesions, and the fact that the disease has occurred endemically suggest a toxic or infectious origin.

Pathology.—The liver is reduced in size, flaccid, and friable. The surface is yellowish-red and mottled. Microscopic examination reveals extensive necrosis of the liver-cells, hemorrhagic extravasations, hematogenous pigmentation, and

occasionally small-celled infiltration. The other organs are usually the seat of fatty and parenchymatous degeneration.

Symptoms.—(1) The initial symptoms are those of catarrhal jaundice. (2) Nervous symptoms (*cholemia*) soon follow; these are severe headache, maniacal delirium, stupor, and coma. (3) The urine is scanty, and usually contains *leucin* and *tyrosin*, bile, albumin, and tube-casts. The excretion of urea is often greatly diminished. (4) The area of hepatic percussion dullness rapidly decreases. (5) Hemorrhages from the mucous membranes and into the skin are common. Fever is usually absent. The disease rarely lasts longer than 2 or 3 weeks. Recovery is extremely rare.

Diagnosis.—In *acute phosphorus-poisoning* acute gastritis precedes the jaundice, the vomitus and stools may be phosphorescent or have the odor of phosphorus, the liver is usually enlarged, and the urine less frequently contains leucin and tyrosin.

In *hypertrophic cirrhosis* the liver is enlarged and often painful, the course is slow, and leucin and tyrosin rarely appear in the urine. In *Weil's disease* the liver is enlarged and high fever is a constant symptom.

Treatment.—This must be symptomatic.

DISEASES OF THE PERITONEUM

ACUTE PERITONITIS

Definition.—An acute inflammation of the peritoneum. The process may be general or localized.

Etiology.—The disease is probably always caused by bacteria, which enter the peritoneum from the adjacent viscera, especially the alimentary canal, from the Fallopian tubes, from external wounds, or directly from the blood. The organisms most frequently found are the *Streptococcus pyogenes*, *Staphylococcus pyogenes*, *Bacillus coli*, pneumococcus, *Bacillus pyocyaneus*, and gonococcus.

Peritonitis may follow—(1) *Perforation* of the peritoneum by an external wound, by rupture of a gastric or intestinal ulcer, by rupture of a suppurating appendix, gall-bladder, or Fallopian tube, or by rupture of a visceral abscess; (2) *extension* of a septic process in adjacent structures—stomach, bowel, gall-bladder, pancreas, uterus, etc.; (3) *traumatism*; (4) *general infections*—septicemia, pneumonia, etc.

Pathology.—The serous surfaces first become red and lusterless; later a serofibrinous, fibrinous, or purulent exudate is formed. Putrid and hemorrhagic exudates are sometimes observed.

Symptoms.—The most prominent symptoms are intense abdominal pain and tenderness. The breathing is shallow and thoracic. To relieve the tension of the abdominal muscles, the patient lies motionless upon his back, with the legs and thighs flexed. The features are pinched, and the expression is anxious. The abdomen is distended, and its walls are rigid. Percussion at first reveals general tympany, but later there may

be dulness in the flanks from the gravitation of the exudate. The temperature is usually moderately high (102° – 104° F.), and the pulse is small, rapid, and “wiry.” The bowels are usually constipated. Vomiting and hiccup are common symptoms. In severe cases collapse speedily ensues, and is indicated by a fall in the temperature, a cold, clammy surface, a rapid feeble pulse, and suppression of urine.

In *localized* peritonitis the constitutional symptoms are less severe. Pain, tenderness, and rigidity are circumscribed. General tympanites is usually absent. Abscess formation is common.

Diagnosis.—*Acute Enteritis.*—In this disease the pain is colicky and less intense; tenderness is much less marked; rigidity is rarely present; there is diarrhea; the constitutional symptoms are not so grave.

Intestinal Obstruction.—Constipation is absolute; vomiting is stercoraceous; fever and abdominal tenderness are less pronounced.

Hysterical Abdomen.—This condition may closely resemble peritonitis. The personal history must be carefully considered. Fever is not usually present, the pulse is not usually rapid and wiry, and when the attention is distracted, the pain may disappear.

Prognosis.—The prognosis in acute diffuse peritonitis is always grave, and in perforative cases it is practically hopeless unless operative treatment is soon instituted. In localized peritonitis the outlook is much more favorable.

Treatment.—The patient should be kept in a semi-sitting (Fowler’s) position, so that the exudation will flow toward the pelvis and away from the diaphragm. All food and liquids by the mouth should be withheld and thirst relieved by enemata of salt solution. Persistent vomiting is best treated by washing out the stomach. Collapse must be combated by the external application of heat, the subcutaneous administration of strychnin, and the intravenous injection of adrenalin in salt solution (1 in 50,000).

Surgical intervention is indicated in the great majority of cases. The operation should be done speedily, the exudation released, and the cause removed with the least possible trauma and handling of the parts. Free drainage should be maintained by Fowler's position. Irrigation of the abdominal cavity is probably better avoided, although some very able surgeons believe that it is often indicated. To maintain the proper degree of tension in the vessels and to favor the elimination of toxins, enteroclysis by the drop (Murphy's) method is invaluable. Morphin in large doses is harmful, but the entire withholding of it is rarely justifiable.

CHRONIC DIFFUSE PERITONITIS

Etiology.—Chronic peritonitis may be a sequel of acute peritonitis. In a few instances it has seemed to have resulted from syphilis. In the vast majority of cases it is tuberculous or cancerous.

Pathology.—The intestines are matted together by adhesions. The omentum is often contracted and greatly thickened. Effusion is usually present, but it varies considerably in amount; it is highly albuminous, and in the tuberculous and cancerous varieties it may be bloody.

Symptoms.—Fever is slight and may be absent. Pain is not severe, and is frequently paroxysmal. There is usually more or less diffuse tenderness. Anemia and emaciation are often pronounced. The abdomen is usually distended, often irregularly, from sacculated effusions, inflated intestinal coils, or the projecting matted omentum. Palpation often detects a friction fremitus and resistant masses or nodules. Percussion yields dulness, varying in extent with the amount of effusion. If the fluid is sacculated, the dulness may be irregularly distributed. Fluctuation can sometimes be elicited. On tapping, the fluid is turbid, rich in albumin, and of high specific gravity (about 1015). In cancerous and tuberculous cases it is frequently bloody.

Diagnosis.—The diagnosis between *tuberculous* and *cancerous* peritonitis is not always easy. The tuberculous form usually occurs in persons under forty, gives rise to less cachexia than the cancerous form, and is frequently associated with tuberculous foci elsewhere, especially in the lung, pleura, testis, or Fallopian tube. In doubtful cases the tuberculin test may be employed or a guinea-pig may be inoculated with the exudate.

Prognosis.—Cancerous peritonitis is invariably fatal. Tuberculous peritonitis, while always grave, not infrequently ends in recovery, especially in children.

Treatment.—In the tuberculous form the general treatment should be that of pulmonary tuberculosis. If the effusion is large, aspiration will be required. Surgical treatment (free incision with washing out of the abdominal cavity with normal salt solution) should be advised in suitable cases.

ASCITES

Definition.—A collection of serous fluid in the peritoneal cavity.

Etiology.—(1) It may result from the causes of general dropsy—heart disease, renal disease, chronic pulmonary emphysema, etc. (2) It is frequent in atrophic cirrhosis of the liver, and is sometimes seen in cancer and syphilis of the liver. (3) It is occasionally caused by thrombosis of the portal vein. (4) It is very common in the various forms of chronic peritonitis. (5) It is sometimes met with in tumors of the abdomen and in extreme enlargement of the spleen. (6) It is rarely due to obstruction or rupture of the thoracic duct (chylous ascites).

Symptoms.—If the infusion is large, a sensation of weight in the abdomen, dyspnea, scanty urination, and edema of the feet may result from pressure.

Physical Signs.—*Inspection.*—The abdomen is distended; the surface is smooth and shiny; the base of the thorax is broadened; the navel is more or less obliterated; the superficial

veins are frequently enlarged; and, when the patient lies in the dorsal position, the flanks bulge.

Palpation may elicit fluctuation, and in the flanks a sense of resistance.

Percussion reveals dulness and resistance in dependent parts, with superincumbent tympany. The dulness is movable and is detected in the flanks when the patient occupies the dorsal position.

Aspiration.—The fluid is usually clear, straw-colored, and albuminous. The specific gravity is from 1010 to 1020. A specific gravity above 1015 points to inflammation. A high percentage of endothelial cells suggests a mechanical causation; of polymorphonuclear leukocytes, subacute inflammation; of lymphocytes, tuberculous infection. In cancerous and tuberculous peritonitis the fluid is sometimes bloody. Occasionally, chylous or chyliform fluid is present.

Diagnosis.—*Tympanites*.—This yields universal hyper-resonance on percussion.

Ovarian Cysts.—The enlargement is at first unilateral. As the intestines are pushed aside, the dulness is anterior and the resonance is in the flanks. Vaginal examination often furnishes important data. The fluid of the cyst has a higher specific gravity (1025).

Distention of the Bladder.—The history, the location of the dulness, and the results of catheterization will make the diagnosis apparent.

Treatment.—Treatment should be directed to the original cause. Hydragogue cathartics and diuretics are sometimes useful. Concentrated saline solutions, compound jalap powder (20 to 40 grains), and elaterium ($\frac{1}{8}$ grain) are the most useful cathartics. Infusion of digitalis (3 to 4 fluidrams), citrated caffein (2 to 3 grains), theobromin (3 grains), theocin (3 grains), potassium citrate or acetate (20 grains), and Guy's pill (see p. 107) are the most reliable diuretics.

R. Potassii citratis..... ʒss
 Infusi digitalis..... ʒvj.—M.
 Sig.—A tablespoonful thrice daily.

If the effusion is large and causes discomfort or great diminution in the quantity of urine, paracentesis should be performed.

Paracentesis Abdominis.—The bladder having been emptied, the patient is placed in a semirecumbent position, and a spot in the median line midway between the umbilicus and the symphysis pubis is anesthetized by means of a block of ice sprinkled with salt. A stout trocar is now introduced with a quick thrust into the abdominal cavity, a rubber tube is attached to the cannula for the purpose of conveying the fluid into a fluid pail placed below the patient's bed, and the trocar is then withdrawn. While the fluid is escaping, a many-tailed bandage is adjusted to the abdomen and gradually tightened. The application of such a binder should never be omitted. It gives support to the relaxed abdominal walls, and tends to prevent syncope and hematemesis. When the fluid ceases to flow, the cannula is removed, and the opening sealed with an antiseptic pad and a few strips of adhesive plaster.

In tuberculosis peritonitis and in cirrhosis of the liver with recurrent ascites surgical treatment sometimes proves successful.

Embolism and Thrombosis of the Mesenteric Vessels.—Occlusion of the mesenteric vessels occurs most frequently in the latter half of life. It may involve the arteries, the veins, or both, and is most commonly due to embolism originating in acute or chronic endocarditis, but it occasionally results from thrombosis due to sclerotic changes in the mesenteric artery or aorta, or to inflammatory changes in the mesenteric vein, occurring in association with morbid processes in the adjacent viscera. Hemorrhagic infarction of the intestine is the usual result. The chief symptoms are acute colicky abdominal pain, vomiting, profuse blood diarrhea, or constipation, abdominal distention, and shock. Peritonitis frequently supervenes. Occlusion of large vessels almost always ends fatally in a few days, unless operation with resection of the bowel is undertaken at an early stage.

DISEASES OF THE KIDNEYS

GENERAL SYMPTOMATOLOGY

THE URINE

Normal urine is a pale, amber-colored fluid, of acid reaction, having a specific gravity of 1015 to 1025, and amounting in quantity to about 50 ounces (1500 c.c.) in twenty-four hours.

Polyuria.—An increased flow of urine.

Temporary polyuria may result from—(1) Excessive ingestion of fluids. (2) Administration of diuretics. (3) Suppression of perspiration. (4) Crises of certain febrile diseases, and certain neurotic manifestations, such as neuralgia and hysteria. (5) Absorption of serous effusions and transudations. (6) Removal of some temporary obstruction in the urinary passages.

Persistent polyuria may result from—(1) Diabetes mellitus. (2) Diabetes insipidus. (3) Chronic glomerulonephritis. (4) Amyloid kidney. (5) Polycystic disease of the kidneys.

Oliguria, diminution in the quantity of urine, occurs in febrile disorders, in conditions causing profuse sweating or diarrhea, in all conditions interfering with the flow of blood through the kidneys, such as valvular heart disease, pulmonary lesions, ascites, etc., and in certain inflammatory or destructive diseases of the kidneys themselves. **Anuria**, or suppression of urine, may occur (1) in acute nephritis and also sometimes in chronic nephritis; (2) after abdominal operations or injuries; (3) in collapse; (4) in hysteria; and (5) as a result of the

mechanical obstruction of the urinary passages by tumors, calculi, enlarged prostate gland, etc.

Urea Excretion.—Normal urine contains about 2 per cent. of urea. The daily output varies considerably, depending upon the diet and other factors. The average amount is between 20 and 40 grams. Estimations of the urea-excretion are of little value unless the total nitrogen intake and the total nitrogen output are also taken into consideration. The quantity of urea is diminished in starvation, in wasting diseases, after prolonged vomiting, and in diseases destroying the liver substance, as acute yellow atrophy. The total excretion of urea is also reduced, though the percentage is often high, in acute and chronic nephritis, especially the glomerular form.

Lithuria.—The amount of uric acid excreted daily in the urine by healthy individuals varies from 0.2 to 1.25 grams, and the ratio of uric acid to urea in health is usually about 1 to 60 or 1 to 70. The uric acid is partly *endogenous*, or derived from the breaking down of the nuclei of the body cells, and partly *exogenous*, derived from the catabolism of the purin bodies of the food (meats, especially glandular organs, caffeine from coffee, etc.). The amount of endogenous uric acid varies in different individuals, but it remains fairly constant for the same individual. A deposit of urates in the urine is no indication of an excess in the daily excretion. Such a deposit may result from increased concentration of the urine, a high degree of acidity, a deficiency of pigment (Klemperer), and even a low atmospheric temperature. Clinically, uratic deposits are common in febrile diseases, in various affections of the stomach and liver, in venous stasis of the organs from diseases of the heart or lungs, in leukemia, and in gout during and after the attack. Observations on uric acid excretion are of little value unless exact quantitative estimations of the daily totals are made and the nature and amount of the foods ingested are taken into consideration.

A sediment of *uric acid* has a reddish-brown color and resembles grains of cayenne pepper. Microscopically, uric

acid appears as reddish-yellow rhombic prisms or lozenge-shaped crystals. Amorphous *urates* form a pinkish sediment, having the appearance of brick dust, and microscopically occur as dark opaque granules. Occasionally urates occur in crystalline form, appearing as needles, dumb-bells, or globules with spiny processes.

Leucinuria and Tyrosinuria.—Leucin and tyrosin are found in the urine chiefly in acute yellow atrophy of the liver and



FIG. 2.—Uric acid and uric acid salts.

phosphorus-poisoning, and, in small quantities, occasionally in the acute infectious diseases.

They may be detected by evaporating a few drops of the urine on a glass slide. Leucin appears in the form of small, round, glistening spheres, resembling fat-drops, but, unlike the latter, they are insoluble in ether. Tyrosin appears in the form of intersecting tufts of fine acicular crystals.

Cystinuria.—Cystin rarely appears in the urine in consequence of an hereditary fault of metabolism, the patient being unable to complete the oxidation of the cystin normally formed in the disintegration of protein. It forms colorless hexagonal crystals freely soluble in ammonia and mineral acids. It is

of clinical importance only in that it is prone to precipitate and form renal or vesical calculi.

Alkaptonuria.—This very rare condition results from a congenital inability to complete the combustion of the aromatic

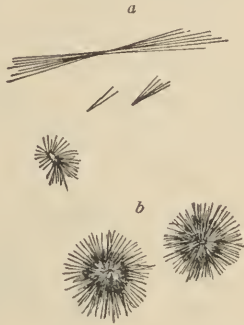


FIG. 3.—*a*, Tyrosin crystals; *b*, leucin crystals.

fractions of proteins. The urine contains an aromatic acid, homogentisic acid, and in consequence turns black on exposure to air, stains the clothing and reduces Fehling's solu-

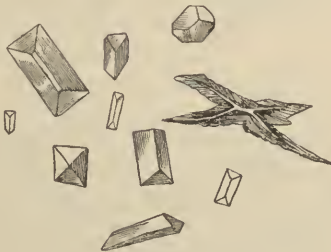


FIG. 4.—Triple phosphate.

tion. Alkaptonuria is harmless, but occasionally leads to blackening of the cartilages, fibrous tissue and skin (*ochronosis*).

Phosphaturia.—Phosphates forming urinary sediments occur in urine that is alkaline, neutral or feebly acid. Both amorphous and crystalline forms are observed. The precipitation of *amorphous earthy phosphates* (calcium and magnesium)

may be effected by warming the urine and adding to it a few drops of liquor ammonia. The precipitate resembles one of albumin, but is distinguished by being soluble in acetic acid. *Crystals of calcium phosphate* are observed in urine that is still feebly acid, but which is about to undergo alkaline fermentation. The crystals are stellate or rod-shaped and are soluble in acetic acid.

Ammoniomagnesium phosphate, or triple phosphate, is formed in decomposing urine and appears as transparent coffin-

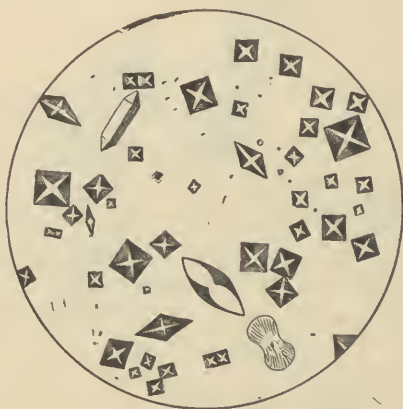


FIG. 5.—Oxalate of lime.

shaped prisms or as feathery stellate forms. The shorter triangular prisms resemble crystals of calcium oxalate but, unlike the latter, are freely soluble in acetic acid. The detection of triple phosphates in freshly voided urine indicates decomposition in the bladder, the result of cystitis.

A deposition of phosphates in the urine does not in itself constitute evidence of excessive elimination. Such a deposition is often due to diminution of the acidity of the urine from various causes. An actual excess of phosphates can be determined only by quantitative methods. Clinically, increase of phosphates occurs in osteomalacia, diabetes, tuberculosis, and many other wasting diseases. It is not rarely associated

with neurasthenia, but whether the relation is one of cause or effect is not known. In certain cases, especially in children, it seems to be the result of some disturbance of calcium metabolism. Finally, cases of polyuria with phosphaturia, resembling diabetes mellitus, have been described (*diabetes phosphaticus*).

Chlorids.—The quantity of these salts in the urine depends largely upon the amount of chlorids in the food. Clinically, the chlorids are diminished in febrile diseases, more especially in pneumonia. In cases of nephritis the retention of chlorids in the tissues constitutes one factor in the causation of dropsy. The quantity of chlorids may be roughly estimated by removing any albumin that may be present, then adding a few drops of a solution of silver nitrate and noting the amount of precipitate obtained.

Oxaluria.—Oxalates are deposited in the urine in the form of calcium oxalate which appears as minute, highly refracting octahedra or as dumb-bells. The amount of oxalate sediment bears no relation to the amount of oxalic acid excreted. Oxalate crystals are especially abundant after certain articles of diet (rhubarb, spinach and other vegetables), and in certain cases of indigestion marked by pronounced neurasthenic symptoms and mental depression.

Cylindruria.—This term is used to designate the occurrence of tube-casts in the urine. Tube casts are cylinders of albuminoid substances formed in uriniferous tubules. They are often composed in part of epithelial cells, blood-cells, or the products of degenerated cells. They appear as:

Hyaline Casts.—These are clear, translucent cylinders, often so pale as to be scarcely visible. They occur in the urine in all forms of nephritis, in congestion of the kidneys, in jaundice, and even in health. They are frequently the only casts present in chronic interstitial nephritis.

Waxy Casts.—These resemble hyaline casts, but they appear more solid and rigid and are more or less yellow. They occur especially in chronic parenchymatous nephritis.

Epithelial Casts.—These are cylinders of epithelial cells or hyaline casts covered with epithelial cells. They occur especially in acute tubular nephritis.

Granular Casts.—These are cylinders covered with the débris of broken-down epithelial cells. They may occur in any form of nephritis.

Fatty Casts.—These are casts studded with oil-drops derived from degenerated epithelium. They occur especially in chronic tubular nephritis.

Blood Casts.—These are cylindric masses of red blood-cells, or, more commonly, hyaline casts studded with red blood-cells. They occur in acute and chronic hemorrhagic nephritis.

Pus and Bacterial Casts.—Casts composed respectively of masses of pus-cells and of bacteria are occasionally met with in suppurative nephritis.

Cylindroids.—These formations may resemble hyaline casts, but they are usually much longer and often taper off at one end to a thread. Moreover, they frequently show constrictions at different points. Their presence is not a proof of nephritis. They often occur in conditions of renal irritation.

Urobilinuria.—Urobilin is formed in the intestines by the action of bacteria on bilirubin. When present in excess, the urine is of a reddish-brown color. An increase of urobilin in the urine may occur—(1) In diseases associated with excessive destruction of red blood-cells, as pernicious anemia and scurvy; (2) after the absorption of hemorrhagic effusions; (3) in acute infectious diseases; (4) in certain liver diseases (cancer, cirrhosis, catarrhal jaundice). The yellowish pigmentation of the skin sometimes observed in cases of urobilinuria is due to bile pigment in the blood and not to the urobilin (see acholuric jaundice, p. 95).

Hematoporphyrinuria.—Hematoporphyrin is a product of the decomposition of hemoglobin. It is hematin deprived of its iron. Large amounts impart to the urine a dark red color. It is found in the urine in a large number of diseases,

and in chronic poisoning by sulphonal and trional and not rarely by veronal.

Glycosuria.—Glucose in the urine.

Causes.—Normal urine contains a trace, but this is not recognizable by the ordinary tests. Clinically, glycosuria is observed—(1) In diabetes mellitus, due to lesions of the islands of Langerhans; (2) after the ingestion of large amounts of sugar (200 to 250 grams of grape-sugar); (3) in certain diseases of the pancreas and other glands having internal secretions, such as the hypophysis, thyroid and adrenals; (4) in injuries or diseases of the nervous system affecting directly or indirectly Claude Bernard's medullary center; (5) after the administration of phloridizin, the resulting glycosuria depending on changes in the renal cells (renal diabetes) and being unaccompanied by a hyperglycemia; (6) not rarely after the administration of ether or chloroform and in poisoning by morphin, anilin derivatives, cyanids, and many other drugs; (7) sometimes in pregnancy; (8) not rarely in the acute infections, grave anemias, and gout.

Qualitative Tests for Glucose.—The copper tests are commonly employed, and depend on the power of glucose in alkaline solution to reduce cupric hydroxid to cuprous hydroxid and cuprous oxid.

Fehling's Test.—As the fluid employed in this test spoils on keeping, it should be freshly prepared when required by mixing in equal proportions the following solutions:

First solution: Dissolve 34.64 grams of pure cupric sulphate in distilled water, and dilute up to 500 c.c.

Second solution: Dissolve 180 grams of pure Rochelle salt and 70 grams of caustic soda in 400 c.c. of distilled water, and heat to boiling; on cooling, make up to 500 c.c. with distilled water.

To about ten minims of each solution in a test-tube add about a fluidram of distilled water, and boil for a few seconds; if the solution remains clear, add the suspected urine drop by drop, and occasionally heat the tube. If sugar is abundant,

a yellowish-red deposit will be produced. If no precipitate falls, continue the addition of the urine until an equal volume has been added, and allow to cool; then if no precipitate falls, sugar is absent.

It must be borne in mind that cupric hydroxid is sometimes reduced by substances in urine other than glucose, such as excess of uric acid or creatinin, alkapton, compound glycuronates following the administration of chloral, salol, turpentine, etc., and other sugars (lactose, pentose). In case of doubt the fermentation test should be tried.

The Phenylhydrazin Test.—Put in a test-tube half filled with water phenylhydrazin (hydrochlorate) 2 grains and sodium acetate 3 grains. Dissolve by heating. Fill the tube with suspected urine, and stand in boiling water for twenty minutes. Then place in cold water. On cooling, yellow, radiating groups of needle-shaped crystals of phenylglucosazon fall, which may be detected under the microscope.

Böttger's Test.—Add to a couple of drams of suspected urine which is free from albumin an equal volume of liquor potassæ and a few grains of subnitrate of bismuth, and boil; if sugar is present, it will reduce the salt of bismuth to black metallic bismuth. Substances containing sulphur, like albumin, yield a similar black precipitate.

The Fermentation Test.—Fill a four-ounce bottle three parts full of urine, and add a fluidram of ordinary yeast or a small portion of compressed yeast; lightly cork, and subject to a temperature of 70° to 80° F. for ten or twelve hours. If sugar is present, fermentation results with the evolution of carbon dioxid, and the specific gravity of the urine falls.

Quantitative Tests.—*Fermentation test:* Employ two bottles of urine, and to the one add the yeast; at the end of twenty-four hours take the specific gravity of each specimen. Every degree lost in the fermented urine indicates a grain of sugar to the fluidounce.

Fehling's Test.—To 1 c.c. of Fehling's solution add 4 c.c. of distilled water, and boil; if the solution still remains clear,

add $\frac{1}{10}$ c.c. of the urine from a graduated pipet, and gently heat. Continue the addition of the urine, little by little, until all blue color has disappeared. If 1 c.c. of urine has been added, it will have contained half of 1 per cent. of sugar. If 2 c.c. are used, it will have contained 14 per cent. If but $\frac{1}{2}$ c.c. is used, it will have contained 1 per cent.

If the specific gravity indicates that the amount of sugar is great, dilute the urine with a definite amount of water, and estimate accordingly (Tyson).

Albuminuria.—Albumin in the urine.

Causes.—It occurs—(1) In all forms of nephritis; (2) in congestion of the kidneys from diseases of the heart, lungs, and liver; (3) in conditions profoundly affecting the blood, as pernicious anemia, leukemia, purpura, and poisoning by many drugs; (4) in acute febrile diseases; (5) often in pregnancy; (6) occasionally in certain persons in health, as in young adults after exertion, after a cold bath, or even upon changing to the upright position (orthostatic albuminuria); (7) when the urine contains pus or blood (accidental albuminuria); (8) in many nervous diseases, as apoplexy, cerebral concussion, tetanus, epilepsy.

Tests for Albumin.—*Heller's Test.*—Pour a small quantity of colorless nitric acid in a test-tube, and allow an equal quantity of filtered urine to trickle from a pipet down the side of the tube and to come in contact with the acid. If albumin is present, a sharply defined white ring is formed at the line of junction.

Turpentine, copaiba, and other oleoresins eliminated in the urine yield similar rings, but the latter are redissolved on the addition of alcohol.

Uric acid produces an undefined pink ring, but it is not exactly at the line of contact, and is redissolved on the application of heat.

Bence-Jones protein produces a ring exactly like that of albumin with cold nitric acid, but heat redissolves it.

Johnson's Test.—Fill a six-inch test-tube two-thirds full of filtered urine, and allow a couple of drams of a clear, saturated solution of picric acid to flow down the side of the tube and to mix with the urine. Turbidity indicates the presence of albumin, and it increases on gently heating the tube near its mouth. Certain substances in the urine, such as the alkaloids, produce a similar turbidity, but this disappears on the application of heat.

Robert's Nitric Magnesium Test.—This test is very delicate and reliable. The test-fluid is made by adding one volume of strong nitric acid to five volumes of a saturated solution of sulphate of magnesium, and is employed in the same manner as nitric acid in Heller's test.

Albumosuria.—True albumose may be found in the urine when large amounts of protein are undergoing autolysis, as during postpartum involution of the uterus, in febrile diseases, in pneumonia during resolution of the exudate, and acute yellow atrophy of the liver. Albumose is not precipitated by heating.

Bence-Jones protein, sometimes regarded as an albumose, appears very early in the urine in the neoplastic affection of the bone marrow known as multiple myeloma and occasionally in the metastatic carcinoma of bones. It is not precipitated by acetic acid, but with strong nitric acid it forms a precipitate, which disappears on heating and reappears on cooling.

Acetonuria.—Acetone is probably derived chiefly from the fats through the intermediary stages first of β -oxybutyric acid and then of diacetic acid. The appearance of these bodies in the urine is spoken of as *ketonuria*. Their production in excessive amount results in *acidosis*. Acetone occurs in the urine—(1) In diabetes mellitus; (2) in starvation, especially carbohydrate starvation; (3) in poisoning with chloroform and phosphorus; (4) in many acute infections; (5) sometimes in the pernicious vomiting of pregnancy; (6) in certain gastrointestinal disturbances, especially of children. In the recurrent vomiting of children the digestive disturbances and the acidosis appear to be the result of a common cause.

Legal's Acetone Test.—To 4 c.c. of urine add a few drops of a strong, freshly prepared solution of sodium nitroprussid and a few drops of acetic acid (to prevent reaction with creatinin); then add sodium hydroxid solution. If acetone be present, the urine acquires a ruby-red color, which soon turns to a purplish red.

Diacetic acid and *beta-oxybutyric acid* are often found in the urine with acetone when the morbid processes responsible for the production of these compounds are very active. The presence of diacetic acid is indicated by the appearance of a Bordeaux-red color when a solution of ferric chlorid is slowly added to urine that has not been boiled. Salicylic compounds give the same reaction, but with these the color change is also observed in urine that has been previously boiled. There is no simple clinical test for beta-oxybutyric acid.

Hematuria.—Blood in the urine. The chief causal conditions are: (1) Traumatism; (2) acute inflammation of any part of the genito-urinary tract—kidneys, bladder, urethra; (3) calculi in the bladder or kidney; (4) congestion or infarction of the kidneys; (5) chronic glomerulonephritis (occasionally copious bleeding occurs); (6) certain general disorders affecting the blood, such as purpura hemorrhagica, scurvy, hemophilia, leukemia, malaria, etc.; (7) tumors or tubercle of the kidney or bladder; (8) varicose veins at the neck of the bladder (occasionally seen in old persons), enlargement of the prostate, benign or malignant; (9) parasites in the genito-urinary tract, such as the *Filaria sanguinis hominis* and *Bilharzia hematobia*.

Diagnosis.—Blood may be recognized by microscopic, spectroscopic or chemical examination. By the unaided eye, blood may be confused with bile, urobilin, or hematoporphyrin.

Heller's Test.—Boil the urine with a solution of caustic potash: phosphates are precipitated, which assume a red color from the freed hematin.

Source of Hemorrhage.—*Urethra.*—The urine first passed is bloody, and the other symptoms point to the urethra.

Bladder.—Bleeding is often at the end of micturition and other symptoms point to the bladder.

Kidney.—The blood is intimately mixed. There may be blood-casts or clots, and the other symptoms point to the kidneys.

In many cases the source of the blood can be determined only by a careful study of the associated symptoms and data obtained from cystoscopy *x*-ray studies, and ureteral catheterization.

Hemoglobinuria.—Hemoglobin appears in the urine as a result of excessive hemolysis. The latter occurs under the following conditions: (1) In certain infections, notably malaria (black-water fever); (2) in poisoning by various drugs, such as potassium chlorate, coal-tar derivatives, etc.; (3) after severe burns; (4) sometimes after the transfusion of blood; (5) sometimes in Raynaud's disease; (6) paroxysmal hemoglobinuria. In this last affection, which is sometimes of syphilitic origin, the attacks are usually precipitated by exposure to cold.

Indicanuria.—Indican, or potassium indoxyl sulphate, is a product of indol derived from the bacterial decomposition of proteids in the intestine. It does not color the urine, but by oxidation it is converted into indigo-blue. It is a constituent of normal urine. It is increased (1) in all conditions which favor putrefaction in the upper bowel, as obstruction in the small intestine, acute and chronic peritonitis, typhoid fever, intestinal catarrh, and obstructive jaundice; (2) conditions associated with the decomposition of pus, as empyema, abscess, and gangrene of the lung.

Tests for Indican.—Mix equal volumes of urine and fuming hydrochloric acid, and with constant shaking, add a fresh, saturated solution of calcium hypochlorite, drop by drop, until the blue color ceases to deepen, then shake with chloroform. The latter dissolves the indigo and separates as a blue liquid, the color of which is more or less deep, according to the amount of indican.

Choluria.—The presence of bile-pigments and bile acids in the urine. It is most marked in obstructive jaundice, but it

may also occur in the non-obstructive form. The urine varies from a greenish-yellow to a dark brown color.

Tests for Bile.—*Gmelin's Test.*—Allow a few drops of urine and a few drops of fuming nitric acid to come together on a white plate. If bile is present, there will be an iridescent play of colors—green, blue, violet, and red—at the line of contact.

Pettenkofer's Test.—Add a few grains of cane-sugar and a drop of sulphuric acid to the suspected urine in a test-tube; heat gently, and if bile acids are present, a violet-red color is produced.

Test for Bile Acid.—Bile acids lessen the surface tension of urine, hence finely powdered sulphur sprinkled on the surface of the urine, instead of floating, sinks if bile acids are present (Hay's test).

Chyluria.—Chyle in the urine. The urine presents a milky appearance. The emulsion of the fat is so complete that microscopic examination rarely reveals distinct oil-globules. Ether dissolves the fat and renders the urine clear. Chylous urine is often slightly pink from the admixture of blood. Chyluria is usually due to obstruction of the lymphatic ducts by the *Filaria sanguinis hominis*, but a non-parasitic form of this condition, obscure in origin, is sometimes observed.

Pyuria.—Pus in the urine. It results (1) from suppurative inflammation of any part of the genito-urinary tract, and (2) from the rupture of abscesses into the tract.

It appears as a dull, greenish-yellow precipitate that is converted into a clear gelatinous mass by the addition of liquor potassæ. It can always be detected by the microscope.

Source.—If the pus is from the kidney, it is intimately mixed with the urine; the latter usually has an acid or neutral reaction, and the associated symptoms point to the kidneys.

If the pus is from the bladder, it is not so intimately mixed with the urine; the latter is often alkaline in reaction, and the associated symptoms point to the bladder.

Ehrlich's Diazo-reaction.—In certain diseases the urine contains aromatic bodies that produce a characteristic color with sulpho-diazobenzol,

Process.—Two solutions should be prepared and kept in separate bottles:

1. Sulphanilic acid.....	5.0	2. Sodium nitrate.....	0.5
Distilled water.....	1000.0	Distilled water	100.0
Hydrochloric acid, pure	50.0		

In order to apply the test, 50 c.c. of No. 1 are added to 1 c.c. of No. 2. The mixture is added to the urine in a test-tube in the proportion of half urine and half mixture. One c.c. of ammonia-water is then added and the test-tube is violently shaken. The reaction is positive only when the resulting froth acquires a rose-red (not brown) color.

The diazo-reaction is commonly present in typhoid fever. Its value in diagnosis is lessened by its frequent occurrence in tuberculosis, measles, pneumonia, and septic diseases.

TESTS OF THE FUNCTIONAL CAPACITY OF THE KIDNEYS

The most important tests are (1) those which deal with the quantitative determination of the excretion of various substances in the urine, especially urea, sodium chlorid, sugar after phloridzin administration, and certain dyes, particularly phenolsulphonephthalein; and (2) those which deal with the quantitative determination of the concentration of certain substances in the blood, especially urea and incoagulable nitrogen. *Cryoscopy*, or the measurement of the molecular concentration of solutions by ascertaining their freezing points, affords information of some value. In health the freezing point of the blood varies from -0.55° C. to -0.57° C., and that of the urine from -1.30° C. to -2.20° C. In nephritis the freezing point of the blood is lowered and that of the urine is raised, the two figures tending to approximate. A smaller output than intake of *sodium chlorid*, if persistent, is usually indicative of renal inadequacy, and leads to salt retention. The latter bears no direct relation to the degree of nitrogen retention or to the development of uremia, but it is, as a rule, accompanied by edema. The urinary chlorid excretions may be estimated by means of an apparatus constructed on the principle of Esbach's albuminometer. The glycosuria which normally follows the subcutaneous injection of *phloridzin* has been found to be slow in appearing or to be wholly wanting in diseases of the kidneys affecting the renal epithelium. Normally, sugar appears in the urine within half an hour and disappears after two to four hours. The results of this test are not entirely reliable in estimating the degree of renal insufficiency. In healthy adults the *total non-protein nitrogen of the blood* ranges between 25 and 40 mg., the *urea nitrogen* between 10 and 18 mg., the *creatinin* between 0.1 and 0.8 mg., and

the *uric acid* between 0.5 and 3.0 mg. per 100 c.c. In chronic nephritis without signs of uremia these values are only moderately increased, but on the approach of uremia there is a marked increase in both the nitrogen and urea concentration. Of the various dyes used to test the excretory capacity of the kidneys, *phenolsulphonephthalein* has been shown to be the most satisfactory. A solution is made containing 6 mg. of the dye to 1 c.c. of salt solution. This is injected into the lumbar muscles under aseptic conditions. Following the injection all the urine excreted is collected at the end of one hour and two hours, is diluted to a suitable amount with alkaline water and its 'phthalein content is estimated by colorimetry. Normally, from 49 to 60 per cent. of the amount injected is recovered in one hour, and from 60 to 80 per cent. in two hours. In severe chronic nephritis the phthalein excretion in two hours usually varies from 40 per cent. to 0. In chronic passive congestion (cardiac disease) the elimination of phthalein, although often considerably reduced in decompensation, is usually rapidly restored with the re-establishment of compensation. This test in conjunction with the estimation of waste nitrogen in the blood, if repeatedly applied, gives a fairly reliable index of the functional capacity of the kidneys, especially in cases of chronic nephritis. It is also of great value in affording an indication of the relative integrity of the two kidneys in cases of unilateral disease if the urine from each kidney can be obtained by means of the segregator or by catheterization of the ureters.

DISEASES OF THE KIDNEYS AND PELVIS OF THE KIDNEY

FLOATING KIDNEY

(Movable Kidney; Nephroptosis)

Definition.—A condition in which the kidney manifests a high degree of mobility.

Etiology.—It is much more frequent in women than in men. Tight lacing, frequent pregnancies, rapid loss of flesh, and over-exertion are reputed causes. Relaxation of the abdominal muscles or mesenteric tissues, congenital or acquired, diminution of the perinephric fat, and preternatural shallowness of the kidney bed are probably the most important etiologic factors.

Symptoms.—The right kidney is the one usually affected, probably from its relation to the kidney, which moves during the respiratory acts. The kidney may be found in any part of the abdomen as a movable tumor, reniform in shape, somewhat tender to touch, and rarely imparting the pulsation of the renal artery. Not infrequently gastroptosis and enterop-tosis coexist.

There may be no subjective symptoms. In many cases, however, there is a sensation of discomfort in the abdomen, accompanied by digestive disturbances, neurasthenic phenomena, and hypochondriasis. Occasionally painful paroxysms occur simulating renal colic (Dietl's crises). These have been attributed by some to engorgement of the kidney from twisting of the renal vessels, and by others to intermittent hydronephrosis.

Diagnosis.—The reniform shape of the tumor, its free mobility, its stationary size, the lessened resistance on percussion over the renal region of the affected side, and the absence of cachexia will serve to diagnose a floating kidney from other abdominal tumors.

Treatment.—In many cases regulation of the diet, the avoidance of undue exertion, the employment of measures intended to increase the body weight and to improve the tone of the abdominal muscles, and the application of a well-fitting supporter will suffice. If the distressing symptoms persist and prove disabling, nephrorrhaphy (stitching the kidney to the posterior abdominal wall) should be considered. Cure or marked relief may be expected to follow operation in the majority of the cases.

HYPEREMIA OF THE KIDNEYS

Active Hyperemia.—Acute congestion of the kidneys marks the early stage of acute nephritis and is due to the same causes (exposure to cold, infectious diseases, overdoses of certain drugs, pregnancy, etc.) as the latter. The kidneys are large, soft, and of deep red color. The blood-vessels are engorged and the epithelium shows evidences of cloudy swelling. *Clinically*, the urine is scanty and contains a small amount of albumin, a few blood corpuscles and epithelia, and a few hyaline and finely granular casts. There is no edema or uremia, and the symptoms soon disappear after the removal of the exciting cause. The treatment is that of the milder forms of acute nephritis.

Passive Hyperemia.—This is usually the result of chronic cardiac incompetence, but it may be due to pressure on the renal veins by a tumor, ascites, or pregnant uterus, and occasionally it is caused by thrombosis or embolism of the renal veins or ascending vena cava. The kidneys are enlarged, firm, of a bluish-red color. In long-standing cases the fibrous tissue is increased and the epithelium more or less degenerated (*cyanotic induration*). *Clinically*, the urine is scanty, turbid

and of high specific gravity. It contains a small amount of albumin, a few blood corpuscles, and a few hyaline casts. Other evidences of venous stasis (dyspnea, enlargement of the liver, edema, etc.) are usually present. Uremia does not occur unless the condition has resulted in nephritis. The treatment is mainly that of the primary disorder. The patient should be kept at rest. The diet should consist chiefly of milk and farinaceous food. Dry cupping is of service. Digitalis (2 to 4 drams of the infusion) is usually indicated. Hydragogue cathartics (salines or compound jalap powder) are useful adjuvants. In the cardiac cases such a capsule as the following may be of service:

R. Pulveris digitalis..... gr. xx —xl
 Theobrominæ..... ʒj
 Extracti nucis vomicæ..... gr. iv.—M.
 Pone in capsulas No. xx.
 SIG.—One capsule three or four times a day.

UREMIA

Definition.—The name applied to a group of symptoms resulting from an auto-intoxication conditioned upon impairment of the renal functions. No known constituent of the urine has thus far been identified as the noxious agent.

Symptoms.—Uremia may occur in any type of nephritis or as the result of any condition causing complete suppression of urine. It may be sudden in its onset and rapid in its course (*acute uremia*), or it may develop slowly and persist for weeks or months (*chronic uremia*). In the acute form the most common manifestations are severe headache, intense restlessness and jactitation, epileptiform convulsions, delirium (usually muttering but occasionally maniacal), coma (generally preceded by convulsions, delirium, or severe headache), dyspnea of an asthmatic or a Cheyne-Stokes type, and persistent vomiting, sometimes associated with hiccup and diarrhea. Transient blindness, unaccompanied by any changes in the fundus of the eye, may also occur.

The more chronic manifestations include dull headache, vertigo, ready mental and physical fatigue, recurrent attacks of nausea and vomiting (often regarded as "bilious attacks"), persistent dyspnea, obstinate insomnia, and various mental derangements. Hemiplegia or monoplegia, usually transitory and probably the result of cerebral edema, is not uncommon. Among the less frequent symptoms may be mentioned muscular cramps, twitching of the limbs, tinnitus aurium, itching of the skin and erythematous eruptions.

The tongue is coated, the breath is foul (ammoniacal or urinous), the urine is generally scanty, or if undiminished of low specific gravity, the temperature is, as a rule, subnormal, although it may be elevated (uremic fever), the arterial tension is usually high, but with advanced myocardial degeneration it may be low, the waste nitrogen of the blood is increased and the 'phthalein elimination is decreased and may be nil. The symptoms of chronic uremia may persist with varying intensity for months, but acute exacerbations are likely to occur at any time.

Asthenic Uremia.—Uremia resulting from complete suppression of urine is marked by insomnia, digestive disturbances, dyspnea, and progressive muscular weakness. Convulsions are absent, and the mind may remain clear almost to the end. Unless the obstruction is removed death supervenes about the tenth or eleventh day.

Diagnosis.—This is based upon the clinical history, the various evidences of nephritis, the state of the arteries and heart, the odor of the breath, and, in doubtful cases, upon the determination of 'phthalein elimination by the kidneys and the amount of non-coagulable nitrogen (nonprotein nitrogen, urea, creatinin, uric acid in the blood. The differentiation of uremic coma from other forms of coma is considered on page 453.

Prognosis.—This is always grave. Recovery is possible, however, even after the most severe symptoms.

Treatment.—The chief indication is to favor elimination. One or two drops of croton oil, diluted with glycerin, or ela-

terium ($\frac{1}{6}$ – $\frac{1}{4}$ gr.) should be given at once. Sweating should be promoted by hot-air or vapor baths. Occasionally the subcutaneous administration of pilocarpin ($\frac{1}{6}$ – $\frac{1}{4}$ gr.) may be desirable, but it should be employed with caution. In robust subjects, especially if coma or convulsions appear, venesection may be practiced, the removal of from 10 to 20 ounces of blood often proving very efficacious. In children a few ounces of blood may be abstracted from the loins by means of wet cups. After the blood has been withdrawn an equal volume of normal saline solution may be given intravenously. Irrigation of the bowel with hot saline solution (105° F.) is also useful. Not rarely lumbar puncture is of value. Convulsions may be controlled by a few whiffs of chloroform, by an enema of chloral (20 to 30 grains), or by injection of morphin ($\frac{1}{4}$ gr.).

Chronic uremia must also be treated by measures intended to lessen the concentration of toxic matters in the blood. The most important of these measures are restrictions in diet, rest, free purgation (salines or compound jalap powder), and copious sweating by means of hot-air or vapor baths. Digitalis, strophanthus, theobromin, etc., are often of service even when arterial tension is high. Morphin is useful in controlling asthmatic seizures, restlessness and insomnia.

ACUTE NEPHRITIS

Acute inflammation of the kidneys may be diffuse or it may involve chiefly the glomeruli (*acute glomerulonephritis*) or the tubules (*acute tubular nephritis*).

Etiology.—The important causes are: (1) General infections, such as scarlet fever, streptococcus septicemia, erysipelas and pneumonia; (2) focal infections, especially tonsillitis; (3) chemical poisoning, as by mercuric chlorid, cantharides, turpentine, etc.; (4) certain auto-intoxications, such as may occur in pregnancy, extensive burns, and generalized eczema. Streptococcus infections, scarlet fever, and cantharides poisoning produce a glomerulonephritis, while pregnancy, cholera, and mercuric chlorid poisoning produce a tubular nephritis.

Pathology.—The kidney is often slightly swollen and of a reddish color or mottled, red and grayish areas intermingling. The capsule strips readily. In glomerulonephritis the capillaries of the tufts are filled with leucocytes or are occluded with hyaline thrombi, and the capsular spaces are filled with blood or inflammatory exudate. In tubular nephritis the tubular epithelium is extensively degenerated and desquamated. In both forms the tissue between the glomeruli and tubules is edematous and infiltrated with wandering cells.

Symptoms.—In many cases the only indications of acute nephritis are the urinary changes and, perhaps, slight edema. In the more severe cases the general symptoms are slight fever; dull lumbar pain; nausea and vomiting; dropsy, usually appearing first in the face; and increasing anemia. Increased arterial tension is often observed and uremia may occur at any period (see p. 140).

The Urine.—The urine is scanty, and occasionally it is suppressed. It is concentrated, of acid reaction, and contains a considerable amount of albumin, epithelial cells, numerous tube-casts (chiefly epithelial, granular, and erythrocytic), and usually more or less blood.

Generally speaking, high blood-pressure, pronounced albuminuria, decided hematuria, a marked increase in the urea of the blood, and a tendency to uremia point to a glomerulonephritis.

Complications.—The most common are pneumonia, inflammation of serous membranes (pleurisy, pericarditis), edema of the lungs, and dilatation of the heart.

Diagnosis.—As the general symptoms are often slight, an exact diagnosis must rest upon an examination of the urine.

Prognosis.—Mild forms of acute tubular nephritis usually subside completely in from a few weeks to a few months. In some instances, however, the disease becomes chronic. Glomerulonephritis may also end in complete recovery, but more frequently recovery is only apparent and the process passes insidiously into a chronic stage. In severe cases of

either type death may occur from pneumonia, pulmonary edema, or uremia.

Treatment.—Absolute rest in bed for several weeks is imperative. Milk, more or less diluted with carbonated water, lime-water, or Vichy, is the best food for a time. Later, cream, gruels, fruit juices and milk-toast may be admitted. Animal broths should be interdicted. Sodium chlorid should be withheld if there is edema.

As there are no direct remedies, the indications are to relieve renal congestion, to lessen the work of the kidneys, and to meet the symptoms as they arise.

At the onset, if there is suppression of urine or much pain, wet or dry cupping over the region of the kidneys is of value. Following the cupping, warm poultices may be applied with advantage, but cantharides, turpentine, or similar counter-irritants should never be used.

The bowels should be kept freely open by means of daily purges, the best being salines and compound jalap powder (30 or 40 grains).

Free sweating is efficacious in promoting excretion by the skin. It may be secured by hot packs, hot-air baths, hot vapor baths, or the hypodermic injection of pilocarpin ($\frac{1}{6}$ grain).

Unirritating diuretics, such as the citrate or acetate of potassium, are of service.

Excessive *dropsy* may require puncture of the swollen parts, a free incision at the outer side of each ankle, or the insertion beneath the skin of fine silver cannulæ (Southey's tubes). *Uremia* demands prompt and energetic treatment (see p. 140). After the acute symptoms have subsided iron may be employed in the form of the carbonate or of Basham's mixture to combat *anemia*.

CHRONIC TUBULAR NEPHRITIS

(The Large White Kidney and Contracted White Kidney)

Etiology.—The disease may be a sequel of acute tubular nephritis, but more frequently it develops insidiously as a

result of chronic infection, general (tuberculosis, malaria, etc.) or focal, of chronic alcoholism, or, possibly, of habitual exposure to cold and wet. In many cases the etiology is obscure. It is most common between the ages of twenty and forty years.

Pathology.—Except in very long-standing cases, the kidney is large, of a yellowish-white color and softer than normal. The capsule is thin and strips readily (*large white kidney*). Late in the disease the kidney is small, pale, and firm. The surface is slightly uneven and the capsule, in places, is adherent (*contracted white kidney*). Microscopically, the chief change is extensive degeneration of the tubular epithelium, the Malpighian bodies, for the most part, being intact. The interstitial tissue is edematous and more or less increased. In the cases far advanced many tubules with their corresponding glomeruli are obliterated and their places filled with fibrous connective tissue (*contracted white kidney*).

Symptoms.—In the milder cases the only indications for an indefinite period may be the presence in the urine of a small amount of albumin and a few pale granular casts.

The more severe forms are characterized by increasing weakness, pallor, digestive disturbances, and edema. The last is usually first noted in the face on rising in the morning, but later it becomes more or less general. Eventually, effusions occur in the serous sacs and the whole body takes on a waxy bloated appearance. There is little tendency to high blood-pressure or to retinitis, the functional capacity of the kidneys as shown by the amount of urea in the blood is fairly good, and uremia is uncommon, although it may develop toward the end. The course of the disease is usually marked by remissions and exacerbations.

The Urine.—In the more severe cases the urine is reduced in amount and of high specific gravity (1018–1025). It contains a considerable quantity of albumin and yields an abundant sediment, which consists chiefly of fatty, granular and hyaline casts, cellular detritus, and fat droplets.

Complications.—Hydrothorax and pulmonary edema are common. Intercurrent acute attacks may at any time be excited by chilling, alcoholic excess, or the ingestion of irritant foods. Pneumonia, pleurisy or pericarditis often develops in the late stages.

Prognosis.—Cure is somewhat exceptional, but in the milder forms life may be prolonged for many years. Copious albuminuria and extensive edema, especially if persistent, are unfavorable signs.

Treatment.—In the more pronounced forms rest, both mental and physical, is an important factor. Flannel or silk should be worn next to the skin. Residence in a warm, equable climate, especially in the winter months, is desirable. Cold bathing should be interdicted, but an occasional hot-air or vapor bath at home is often useful.

Foci of infection should be sought for, and if found removed. The diet should be simple, but not too spare, even in regard to the proteins. Alcohol is harmful and condiments, including salt, should be restricted to a minimum. In severe cases an exclusive milk diet may for a time be advantageous. The bowels must be kept free. The organic salts of potassium, caffein, and theobromin may be of service in increasing the urinary output. Myocardial insufficiency requires rest and effective digitalis therapy. Basham's mixture should not be used unless there is actual anemia and even then its effects should be carefully observed.

Extensive and persistent dropsy may yield to rest, restriction of fluids, a salt-free diet, the use of hydragogue cathartics (Epsom salt, compound jalap powder, elaterium), of diuretics, especially caffein, theobromin or theocin, and of diaphoretic measures, particularly hot-air baths or hot packs. Pilocarpin should, as a rule, be avoided. The following diuretic mixture is often useful:

℞. Potassii acetatis..... ʒij
 Infusi scoparii.....
 Infusi digitalis..... āā fʒiij.—M.

Sig.—A tablespoonful three or four times a day.

Not rarely, the edema is so pronounced or so situated that operative measures (aspiration of serous sacs, puncture of the scrotum, incisions near the ankles, insertion of Southey's tubes) become necessary. Uremia will require special treatment (see p. 141), and acute exacerbations should be treated as primary attacks of acute nephritis.

CHRONIC GLOMERULONEPHRITIS

Etiology.—The disease may develop out of acute glomerulonephritis, but in the majority of cases it is produced gradually by septic infection, chiefly with streptococci which have emanated from a primary focus in the tonsils or elsewhere. The etiology, however, is often obscure and it is likely that other irritants arising from without or from within the body may also be a causal factor. The disease is most common between the ages of twenty and forty years.

Pathology.—At an early period the kidney is somewhat enlarged, opaque, and of a grayish or, as a result of hemorrhagic pigmentation, reddish-gray color. The capsule strips readily. In advanced cases the organ is small, firm and red, its capsule is more or less adherent, and its surface is granular (secondary contracted kidney).

Microscopically, the conspicuous changes are in the glomeruli, although the tubular epithelium is always degenerated to some extent, and the interstitial tissue is always increased. In the early stages the glomerular changes are similar to those occurring in acute glomerulonephritis (see p. 143). In long-standing cases many of the glomeruli are shrunken and transformed into hyaline or fibrous globules and many of the tubules are obliterated.

Symptoms.—Loss of flesh and strength and increasing pallor are often the earliest indications. Digestive disturbances are very common. Cardiac symptoms, especially dyspnea on exertion and palpitation, are sometimes prominent features. Headache, dizziness, and insomnia often result from the disturbed circulation or from uremia. Impairment of vision from

albuminuric retinitis is observed more frequently than in any other form of nephritis and is of serious import. Dropsy is somewhat exceptional, although edema sometimes appears late in the disease in consequence of cardiac insufficiency. Uremia is of frequent occurrence.

The blood-pressure is high, a systolic figure of 200 or 220 not being unusual. The aortic second sound is accentuated, the heart is enlarged especially to the left, and the arteries are thickened and tortuous.

The *urine* is abundant (2000–4000 c.c.), the polyuria being especially marked at night. The specific gravity is low and somewhat definitely fixed at from 1013 to 1010; albuminuria is slight and at times may be absent; and casts are usually few in number and for the most part hyaline or faintly granular. Hematuria is sometimes noted.

In the later stages of the disease the functional capacity of the kidneys is impaired, as shown by the phthalein output in the urine and the accumulation of nitrogenous bodies (urea, creatinin, uric acid, etc.) in the blood.

Complications.—The most common complications are albuminuric retinitis, cerebral hemorrhage, dilatation of the heart, pulmonary edema, uremia, inflammation of serous membranes (pleurisy, pericarditis) and pneumonia.

Prognosis.—In mild cases the patient's condition may remain fairly good for a number of years, and even a cure is possible if the underlying cause can be found and removed. In well-developed cases the outlook is unfavorable and the duration shorter than that of chronic tubular nephritis. The number and type of casts and the amount of albumin in the urine are of no value as guides to prognosis.

Treatment.—This in general is similar to that of chronic tubular nephritis. Important features are the removal of any focus of infection, or any toxic condition that may be etiologically related to the nephritis, regulation of the diet, especially as regards proteins, and the avoidance of mental and physical strain, overeating, use of alcohol, chilling of the body and all

other factors that may increase the blood-pressure or overburden the heart. If the arterial tension is excessive it may be brought within bounds by rest, restriction of diet, free catharsis, and, if necessary, the abstraction of blood. Nitrites should be reserved for emergency and used only for short periods. If used too freely they may precipitate uremia. Myocardial insufficiency should be combated with rest and digitalis. The latter is indicated irrespective of the degree of arterial hypertension. Impending uremia may sometimes be averted by absolute rest, restriction of the diet to milk, free purgation, hot packs, and the use of digitalis and caffeine, theobromin or theocin.

ARTERIOSCLEROTIC CONTRACTED KIDNEY

The above term is applied to a form of renal sclerosis resulting from widespread arteriosclerosis. It is to be distinguished from the secondary renal sclerosis occurring in protracted cases of glomerulonephritis and also the atrophic kidney of old age. All three of these conditions have been designated *chronic interstitial nephritis*.

Pathology.—The gross appearance of the kidney is similar to that observed in protracted cases of glomerulonephritis. Microscopically, the chief findings are a great increase of fibrous tissue, sclerosis of the vessels and degenerative atrophy of the tubules and glomeruli.

Symptoms.—The symptoms are largely those of general arteriosclerosis with vascular hypertension, the renal condition being more or less incidental. There is some tendency to polyuria; the specific gravity of the urine is usually somewhat low, but not so definitely fixed as in glomerulonephritis; a small amount of albumin and a few granular or hyaline casts are found in the urine regularly or at intervals; the functional capacity of the kidneys remains good until a very late stage of the disease; there is little tendency to uremia or retinitis; and the general health is often fairly well preserved for a long period, not rarely ten or fifteen years.

Death is usually due to myocardial insufficiency, an arterial accident, angina pectoris, or intercurrent infection, especially pneumonia.

Treatment.—The treatment is mainly that of the primary cardiovascular disease.

AMYLOID DEGENERATION OF THE KIDNEY

(Waxy Kidney; Lardaceous Kidney)

Etiology.—It occurs in prolonged suppurative diseases, especially of bones, in tuberculosis, syphilis, and cachectic states.

Pathology.—The kidney is enlarged, firm, and pale, and on section may present a waxy, translucent appearance. The amyloid areas are colored mahogany brown by the application of Lugol's solution to the cut surface. Other organs, especially the liver and spleen, usually share in the degenerative process.

On microscopic examination the walls of the blood-vessels, especially of those of the Malpighian bodies, are found thickened and infiltrated with a homogeneous wax-like material that turns pink when treated with gentian-violet. The epithelium is often fatty.

Symptoms.—Most patients appear badly nourished and anemic. Dropsy is present in many cases. The liver and spleen are usually enlarged from the same cause. Uremia is are.

The Urine.—The urine is increased in quantity, is rich in albumin, and is of low specific gravity. Microscopically, it contains hyaline and waxy tube casts and degenerated epithelium.

Diagnosis.—This is based upon the history, the enlargement of the liver and spleen, and the polyuria with marked albuminuria.

Prognosis.—In the majority of cases the prognosis is very grave. In the early stages an arrest of the process is not impossible if the original disease can be cured.

Treatment.—The treatment is chiefly that of the primary disease. In other respects it must be purely hygienic, dietetic, and symptomatic.

PYELITIS AND PYELONEPHRITIS

Inflammation of the pelvis of the kidney (*pyelitis*) and inflammation of the kidney substance frequently coexist, one condition causing the other. The conjoint affection, which is known as *pyelonephritis*, is often suppurative. Pyelitis may be catarrhal, suppurative or ulcerative; unilateral or bilateral; and acute or chronic.

Etiology.—Pyelitis or pyelonephritis is always the result of infection reaching the kidney or its pelvis from the lower urinary tract (urogenous infection), from the blood stream (hematogenous infection), or directly by contiguity from some adjacent structure (colon, appendix, etc.), or by means of a wound. The bacteria most frequently concerned in the process are the colon bacillus, the pus cocci, the typhoid bacillus, and the *Bacillus proteus vulgaris*. Among the predisposing causes of infection may be mentioned general infections, trauma, exposure to cold, gastro-intestinal diseases, excretion of irritating drugs, calculi in the bladder or renal pelvis, cystitis, and especially obstruction of the urinary passages with stasis of urine (urethral stricture, prostatic hypertrophy, pelvic tumors, pregnancy, etc.).

Pathology.—The kidney is pale, more or less enlarged and edematous. In the obstructive cases the pelvis is distended at the expense of the kidney substance. The lining membrane is injected, covered with mucus, and not rarely ulcerated. In suppurative pyelonephritis the renal substance may be riddled with miliary abscesses or may be almost completely destroyed, the whole organ being transformed into a large multilocular pus sac (*pyonephrosis*). In long-standing cases the pus may undergo inspissation and calcification, forming putty-like or mortar-like masses.

Symptoms.—The symptoms are often vague and overshadowed by those of the primary disease. Constitutional disturbances, varying markedly in degree, are usually present in the acute cases and in exacerbations of chronic cases, the most common being chills, fever (100° to 104° or more), sweats, leukocytosis, digestive disturbances, and loss of weight and strength. In addition there may be local pain, tenderness in the kidney region, frequent micturition, and in the event of pyonephrosis a smooth rounded tumor. The urine contains pus, mucus, epithelial cells, blood cells, bacteria, albumin, and in pyelonephritis casts of various forms. A larger amount of albumin than can be accounted for by the pus and blood suggests involvement of the kidney substance. The amount of urine varies. In acute cases it is usually diminished and in chronic cases, unless both kidneys are seriously affected, it is generally increased. In acute cases the reaction of the urine is usually acid, but in the chronic forms it is often neutral or alkaline.

Diagnosis.—An exact diagnosis can almost always be made from the history of the case, a thorough physical examination, careful urinalysis, including cultures (catheterized specimens), functional tests (see p. 136), cystoscopy, ureteral catheterization and radiography. In *cystitis* pain is referred to the hypogastric region, there is pronounced dysuria, the urine is more likely to be alkaline in reaction than acid, and lumbar symptoms and signs are absent.

Calculus Pyelitis.—Sharp pain, increased by jarring movements, and reflected down the ureters, and the presence of much blood in the urine point to calculous pyelitis. Ureteral catheterization and x-rays are valuable aids in diagnosis.

Renal tuberculosis may be recognized by the history, the presence of tuberculous foci in other organs, and the discovery of tubercle bacilli in the urine. The tuberculin test may also aid in the diagnosis.

Prognosis.—Mild acute attacks of pyelitis usually end in recovery. In acute pyelonephritis much depends upon the

character of the infection, the extent of the renal involvement, and whether the disease is unilateral or bilateral. Chronic pyelitis and chronic pyelonephritis may last for years. The most grave cases of the latter are those in which both kidneys are involved as a result of obstruction and in which the urinary reaction is alkaline.

Treatment.—In acute cases the important measures are removal of the cause, if possible, rest in bed, the application of cups or warm compresses over the kidney region, a simple unirritating diet, the liberal use of diluents, free catharsis, preferably by means of copious warm enemas, and the administration of antiseptics, such as hexamethylenamin (5–10 grains three or four times a day), and, if necessary remedies to modify the reaction of the urine, such as potassium citrate in one case and benzoic acid in another. The pyelitis of early childhood, which is usually caused by the colon bacillus, often yields to alkaline treatment (15 grains of potassium citrate 4 times a day). If pain is severe morphin will usually be required. In protracted cases autogenous vaccines are sometimes useful. In chronic pyelonephritis special attention must be paid to the underlying condition (stricture, prostatic infection). Surgical intervention is often required.

NEPHROLITHIASIS

(Renal Calculus; Gravel)

Definition.—Renal calculi are concretions formed in the kidney by the precipitation of various solid constituents of the urine.

Etiology.—The disease is more common in males than in females. Heredity and sedentary habits are given as predisposing causes. The formation of stones is favored by the presence in the urine of any sparingly soluble substance in excess. Mucus, blood, pus, or epithelium may furnish the nucleus.

Pathology.—The size of renal concretions varies from that of coarse sand (“gravel”) to that of a large bean. Several

stones are often present and in from 10 to 15 per cent. of the cases both kidneys are affected. The most common forms are those composed of—(1) Uric acid and its compounds; (2) oxalate of calcium; (3) phosphate of calcium and of ammonio-magnesium phosphate. Stones composed of xanthin and cystin are rare.

Uric acid stones are the most common. They are usually smooth, of a reddish-brown color, and comparatively hard. *Oxalate calculi* are very hard, of a dark brown color, and uneven (mulberry calculi). *Phosphatic calculi* are grayish-white in color, soft, and mortar-like.

Events.—(1) Small particles are frequently passed without serious disturbance. (2) Larger concretions may be extruded with intense pain (*renal colic*). (3) Calculi may remain in the pelvis and excite pyelitis or pyelonephritis. (4) They may obstruct the ureter and cause hydronephrosis or pyonephrosis, or if the obstruction is complete, atrophy of the kidney.

Symptoms.—*Pain and tenderness* in the kidney region are common symptoms. The pain is aggravated by rough motion, and tends to radiate along the ureter. *Irritability of the bladder* is sometimes a prominent feature. The urine frequently contains *blood, pus, epithelium*, and *crystals* indicating the nature of the stone.

Symptoms of Sepsis.—Irregular fever, chills, sweats, leucocytosis, and pallor often mark the occurrence of suppurative pyelitis. *Renal colic* is excited by the entrance of the stone into the ureter. It is characterized by intense pain radiating from the kidney downward into the groin, thigh, and testicle. The testicle is often retracted. There are often nausea, vomiting, and collapse. After such an attack the urine may contain blood or particles of stone. *Anuria* is one of the most serious complications.

Diagnosis.—Radiography, cystoscopy, and ureteral catheterization are valuable aids to diagnosis. In the differential diagnosis one must exclude biliary colic, intestinal colic, Dietl's crises, tumor of the kidney, tuberculosis of the kidney,

prostatic disease (hypertrophy, cancer, chronic prostatitis), vesical calculus, disease of the spine, and lumbago.

Prognosis.—In uncomplicated cases the prognosis is good under appropriate treatment.

Treatment.—This should be directed to the underlying diathesis. In cases of uric-acid calculi alkalis and alkaline mineral waters are useful. A quart of water containing 40 grains of potassium bicarbonate and 20 grains of lithium citrate may be taken daily. Special remedies, like piperazin, lycetol, and urosin, have been recommended as solvents, but they are of doubtful value. If phosphatic calculi are present, benzoic or boric acid (5 to 15 grains thrice daily) may be employed in a similar manner.

Operation (nephrolithotomy, pyelotomy, or nephrectomy) is urgently demanded if the attacks of renal colic occur with such frequency as to prove disabling, if there are evidences of severe pyelitis, or if there is reason to believe that the calculus has become permanently impacted in the ureter.

Renal Colic.—The indications are to relieve the pain and to relax the spasm. This is best accomplished by hypodermic injections of morphin and atropin, coupled with hot baths or local applications—hot poultices or fomentations. If the pain is extreme, it may be desirable to administer chloroform. Simple diluents should be given freely. In mild attacks full doses of acetphenetidin or antipyrin, with an abundant supply of hot drinks, may suffice.

HYDRONEPHROSIS

Definition.—Overdistention of the renal pelvis with urinous fluid, the result of mechanical obstruction.

Etiology.—The chief causes are: (1) Congenital stricture, twisting or other anomaly of the ureter. (2) Impaction of a calculus in the ureter. (3) Abdominal tumors compressing the ureter. (4) Tumors growing within the urinary passages. (5) An inflammatory stricture of the ureter or urethra. (6) Compression of the urethra by an enlarged prostate.

Pathology.—The pelvis reveals all grades of distention. In extreme cases it may contain several quarts of clear or turbid fluid, of low specific gravity, and exhibiting traces of the normal urinary ingredients. There is more or less atrophy of the renal tissue. Both kidneys are usually affected when the obstruction is in the lower urinary tract.

Symptoms.—Slight distention yields no symptoms. In other cases a tumor slowly develops in the region of the affected kidney. Not rarely the tumor periodically disappears and reappears, the disappearance being associated with the passage of a large amount of urine (intermittent hydronephrosis). Large cysts are often painful.

Diagnosis.—This should be based upon the history and the exclusion of other abdominal enlargements.

Pyonephrosis may usually be excluded by the absence of purulent urine and symptoms of sepsis.

Ovarian cysts enlarge upward from the pelvis, are more mobile, more superficial, less likely to be overlaid by intestine, and more likely to displace the uterus.

Prognosis.—If the cyst is unilateral and of no great size, life may be indefinitely prolonged. The disease may end fatally in consequence of rupture into the peritoneum, secondary pyonephrosis, or, if bilateral, of uremia.

Treatment.—Large accumulations will demand surgical treatment; small ones should not be disturbed.

TUBERCULOSIS OF THE KIDNEY

Etiology.—The etiology of renal tuberculosis is that of tuberculosis in general. The infection may be hemogenic, lymphogenic, or urogenic. Males are more frequently attacked than females. The majority of cases are encountered between the ages of twenty and forty years.

Pathology.—Two forms of renal tuberculosis have been recognized—the miliary and the caseous. The former is nearly always bilateral, is an acute process, and is generally unmistakably secondary to tuberculosis elsewhere in the body.

The caseous variety runs a chronic course; it usually begins as a unilateral affection, although the other organ is commonly ultimately involved, and a primary focus may or may not be apparent in some other structure. The ureter and bladder are often affected, and in this event either the kidney or the bladder may have been the starting point of the infection in the urinary tract.

Symptoms.—The chief symptoms are: pain in the lumbar region, usually dull, but sometimes sharp like that of renal colic; tenderness on pressure; frequency of urination; dysuria; slight, irregular fever, and more or less cachexia. The urine is usually acid in reaction, and may contain pus, blood, albumin, tubercle bacilli, cheesy particles, and débris. Tube casts are rarely found. In many cases some enlargement of the affected organ can be detected by bimanual palpation.

Diagnosis.—*Calculous Pyeletis.*—In this condition pain is usually more severe and more likely to be affected by movement. Hematuria is more profuse, and is often excited by exertion. Cachexia is not so marked, and there are no tubercle bacilli in the urine. The tuberculin test, x-rays, or catheterization of the ureter may decide the diagnosis.

Tumors of the kidney grow rapidly and usually reach a very large size, produce copious hematuria, but rarely cause pyuria, and are often painless.

Prognosis.—This is always grave. Without intervention the duration is from a few months to several years.

Treatment.—When the renal disease appears to be primary and unilateral, and the patient's strength will permit, nephrectomy should be recommended. The operative mortality in aseptic cases is less than 5 per cent. Complete cure is the rule in the early stages. In advanced cases the treatment must, of necessity, be palliative.

POLYCYSTIC DISEASE AND MALIGNANT TUMORS OF THE KIDNEY

Polycystic Disease.—In this condition, which is usually congenital but not rarely latent until adult life, the kidney is greatly enlarged and

composed of a vast number of cysts varying in size from that of a pea to that of a walnut. The disease is generally bilateral and probably depends upon a defect of development. Clinically it is characterized by the occurrence of tumor masses in both renal regions, intermittent hematuria, and the urinary and cardiovascular phenomena of chronic glomerulonephritis. Operation (unilateral nephrectomy or puncture of the cysts through an incision in the loin) should be considered when there is persistent hematuria, suppuration, or mechanical obstruction.

Malignant Tumors.—The primary malignant growths of the kidney comprise sarcoma, hypernephroma, and carcinoma. Sarcoma is the most common tumor in children and hypernephroma is the most common tumor in adults. Carcinoma is comparatively rare. The chief symptoms are progressive emaciation, cachexia, hematuria (often profuse), and the occurrence of a tumor springing from the lumbar region, overlaid by the colon, sometimes movable in respiration and by palpation, and usually separated from the liver or spleen above by an area of resonance. Pain is inconstant and the urine, apart from the hematuria, often affords no indications. Metastasis is frequently observed, hypernephromas showing a special tendency to involve the lungs and bones and to invade the renal vein and vena cava. Except for intolerable pain or uncontrollable bleeding, operation is contraindicated if metastases are found.

DISEASES OF THE BLOOD AND THE BLOOD-FORMING ORGANS

NORMAL BLOOD

In health the blood amounts to about one-thirteenth of the body-weight. Normally there are approximately 5,000,000 red blood-corpuscles in the cubic millimeter. This number is temporarily diminished during menstruation, gestation, lactation, and fatigue, and after the ingestion of much fluid. Fasting and profuse sweating increase the number of red cells by concentrating the blood. In the first few days of life the number in a cubic millimeter may be 7,000,000 to 8,000,000. In high altitudes the number is also increased. There are from 5000 to 10,000 white cells in the cubic millimeter, the ratio of white to red cells being about 1 to 500. The number of blood-plates is from 200,000 to 350,000.

EXAMINATION OF THE BLOOD

Estimation of Hemoglobin.—The percentage of hemoglobin may be determined by either Fleischl's or Tallqvist's hemoglobinometer, although the former is preferable.

Fleischl's instrument consists of a metal stand with a circular aperture in the center, under which is placed a reflector made of plaster-of-Paris. The aperture is fitted with a small cell having a glass bottom, and divided into two equal compartments. A graduated wedge of colored glass is employed as a standard, the 100 on the scale being intended to represent the percentage of hemoglobin in normal blood. This wedge of glass is so arranged that when moved under the stand, one compartment of the cell will receive white light from the re-

flector, and the other, red light from the tinted glass. A small capillary tube is held over a drop of blood until filled, and is then washed in one of the compartments of the cell, in which has been previously placed some distilled water. Both compartments are then equally filled with water, and the wedge of glass is moved by means of a thumb-screw until the tints in the two chambers are exactly the same, when the percentage of hemoglobin may be read off.

In the examination it is necessary to use artificial light. The 100 mark on the scale, which is intended to represent the percentage of hemoglobin in normal blood, is too high for the average person, 85 or 90 per cent. rarely being exceeded.

In *Tallqvist's method* the color of a drop of undiluted blood soaked into a standard filter paper is compared with tints ranging from 10 per cent. to 100 per cent. on a prepared color scale. This method is very simple and sufficiently accurate for practical purposes.

Enumeration of Red Blood-corpuscles.—The best instrument for blood counting is the hemocytometer of Thoma-Zeiss. This consists of a glass slide in the center of which is a cell $\frac{1}{10}$ mm. in depth. The floor of the cell is divided into 400 small squares, each of which has an area of $\frac{1}{400}$ square millimeter. These small squares are grouped into sets of 16 by a series of additional vertical and horizontal lines bisecting each fifth column of squares. As the depth of the cell from the cover-glass is $\frac{1}{10}$ mm., the cubic contents of each small square is $\frac{1}{4000}$ cm.

The blood is mixed in a *mélangeur*—that is, a capillary tube one extremity of which is blown into a bulb having a capacity of 100 cm. The *mélangeur* is marked at 0.5, 1, and 101. A drop of blood issuing from a prick of the finger is sucked cautiously into the tube exactly to the mark 0.5. The point of the tube is quickly wiped dry and immersed in the diluting fluid (2.5 per cent. solution of potassium bichromate or Toison's fluid),¹ which is drawn up to the 101 mark. The instru-

¹ Methyl-violet, 5 B, 0.025 gm.; sodium chlorid, 1 gm.; pure sodium sulphate, 8 gm.; neutral glycerin, 30 c.c.; distilled water, 160 c.c.

ment is now shaken to secure diffusion of the blood. The diluting fluid remaining in the stem of the mélangeur is now blown out, and a drop of the mixture placed upon the middle of the bottom of the divided cell. The drop in the cell should be free from bubbles and the cover-glass so adjusted that concentric rings of color appear at the points of contact between the cover-glass and the glass plate. Before counting, a few minutes should be allowed for the corpuscles to settle to the bottom of the cell. The number of corpuscles is then counted in 400 small squares. To avoid repetition in counting, corpuscles on the upper and left boundary-lines should be counted, but those on the lower and right boundary-lines should be disregarded. The number of corpuscles in each cubic millimeter of blood is determined by multiplying the number of corpuscles counted by the degree of dilution (200) and again by the cubic contents of each square (4000), and then dividing the product by the number of squares counted (400). Thus, if 2000 corpuscles were counted in 400 squares, the number of corpuscles in each cubic millimeter would be 4,000,000:

$$\frac{2000 \times 200 \times 4000}{400} = 4,000,000$$

After using, the mélangeur should be carefully washed in water, alcohol, and ether.

Enumeration of White Blood-corpuscles.—For counting the white blood-cells a mélangeur should be used which allows a dilution in the proportion of 1:10 and an aqueous 0.5 per cent. solution of acetic acid, to which may be added a little methyl-violet, should be selected as a diluting fluid. The red cells disappear in this solution, and the white cells remain and are readily counted. The latter should be counted in 800 small squares. The number of leukocytes in each cubic millimeter is then determined by multiplying the whole number counted by 4000, and again by 10, and dividing by 800.

The Study of the White Blood-corpuscles.—In normal blood the following forms of leukocytes may be recognized.

1. *Small Lymphocytes*.—These are small cells about the size of the red blood-corpuscles. The nucleus is very large and spheric, and stains intensely with basic stains (methylene-blue). With Ehrlich's triacid mixture the nucleus is pale. The narrow rim of protoplasm surrounding the nucleus is non-granular (hyaline). Small lymphocytes constitute from 20 to 30 per cent. of all leukocytes.

2. *Large Lymphocytes*.—These cells resemble those just described, but are considerably larger. The nucleus is relatively not so large, and stains less deeply. In some forms the nucleus is more or less bent or indented (transitional leukocytes). Normally, large lymphocytes make up from 5 to 10 per cent. of the blood-corpuscles.

Polymorphonuclear Neutrophiles.—These cells are somewhat smaller than large lymphocytes and are actively ameboid. The nucleus appears to be divided into two or more segments and stains deeply. The protoplasm is studded with fine granules, which do not stain well with either simple basic stains (methylene-blue) or simple acid stains (eosin). With Ehrlich's triacid mixture the granules are colored violet and the protoplasm pale pink. Neutrophiles make up from 60 to 70 per cent. of the white cells of normal blood.

Eosinophiles.—These resemble the polymorphonuclear neutrophiles, but are more irregular in outline, and the granules are larger, more highly refractive, more loosely attached, and have a special affinity for acid stains (eosin). Eosinophiles make up from 1 to 4 per cent. of the leukocytes.

Mast-cells (Basophiles).—These cells have a lobulated nucleus. The protoplasm is studded with granules having an intensely basic reaction. These granules remain unstained with Ehrlich's triacid mixture, but with methylene-blue they stain deep blue. Mast-cells are only occasionally encountered in normal blood.

In disease, additional forms are sometimes found. Thus, in leukemia large cells are found which are non-ameboid and which have a single round or oval nucleus imbedded in proto-

plasm containing neutrophilic granules. These have been termed *myelocytes*.

With the aid of a $\frac{1}{12}$ -inch oil-immersion lens large and small leukocytes can readily be distinguished in preparations of fresh blood, but to study satisfactorily the various forms it is necessary to dry and then stain the specimen.

PLETHORA

An increase in the whole quantity of blood. It is very doubtful whether such a condition can be more than transitory.

HYDREMIA

An excess of water in the blood. As a loss of corpuscular elements is generally replaced by the addition of water extracted from the tissues, most anemias are associated with hydremia. The condition is more marked in general dropsy. Temporary hydremia is produced by the excessive ingestion of fluids.

ANHYDREMIA

A deficiency of fluid in the blood. It is observed in starvation, immediately after hemorrhage, and after copious discharges, as in cholera.

MELANEMIA

A condition in which free pigment granules occur in the blood. It is met with in malaria and certain other fevers, and occasionally in melanosarcoma and in Addison's disease. The pigment may be found in the plasma or in the leukocytes.

POLYCYTHEMIA

Polycythemia, or an increase in the number of red cells, is an apparent condition in blood taken from cyanosed parts. It is observed temporarily in the new-born, in recovery from certain anemias, after transfusion of blood, and in blood concentrated by excessive discharges. Marked polycythemia is

sometimes produced by residence in high altitudes and by certain poisons, such as phosphorus and carbon monoxid. It also occurs in the condition known as erythremia.

MICROCYTOSIS AND MACROCYTOSIS

Microcytosis and macrocytosis are conditions in which the red cells are respectively diminished and increased in size. They may occur in any form of severe anemia, but the latter is especially marked in pernicious anemia.

POIKILOCYTOSIS

Poikilocytosis, a condition in which the red cells are irregular in shape, is common in grave anemias, especially pernicious anemia

NUCLEATED RED CELLS

Nucleated red cells (erythroblasts) are divided into three forms—normoblasts, macroblasts, and microblasts. The first resemble in size and color a normal red cell, the second are larger, and the third smaller. Nucleated red cells are not found normally in the circulating blood; they are present, however, in grave forms of anemia. A predominance of megaloblasts is very suggestive of pernicious anemia.

LEUKOCYTOSIS

Leukocytosis, or hyperleukocytosis, is an increase in the number of white cells, especially of the polymorphonuclear forms, in the peripheral blood. It occurs *physiologically* in the new-born, during digestion, in pregnancy and parturition, and after heavy exertion, cold bathing, and massage.

Pathologic leukocytosis is observed in the following conditions: (1) Inflammation. There is an absolute increase in the polymorphonuclear neutrophiles. (2) Infectious diseases. Most infections excite leukocytosis, but the condition is usually wanting in typhoid fever, malaria, measles, influenza and miliary tuberculosis. In any infection in which the toxemia is

intense or the resistance of the individual is slight leukocytosis may also be wanting. (3) Malignant disease, if sufficiently extensive. (4) After hemorrhage. (5) After the administration of certain drugs, such as pilocarpin, antipyrin, salicylates, ergotin, and tuberculin. (6) In certain autointoxications, such as gout and uremia.

EOSINOPHILIA

An increase in the number of eosinophiles in the blood occurs in (1) helminthiasis (trichiniasis, uncinariasis, hydatid disease, etc.); (2) bronchial asthma; (3) certain skin diseases, notably eczema, pemphigus, psoriasis, and urticaria; (4) splenomedullary leukemia; (5) many postfebrile conditions; (6) anaphylactic intoxication.

LEUKOPENIA, OR HYPOLEUKOCYTOSIS

Leukopenia, or hypoleukocytosis, is the name applied to a deficiency in the number of leukocytes. It occurs in certain infections, particularly in those that do not produce leukocytosis, such as typhoid fever, malaria, and miliary tuberculosis; also in pernicious anemia and inanition.

LIPEMIA

Lipemia, the presence in the blood of minute fat-globules, may be noted in health. Abnormal quantities of fat may be found in the blood in diabetes, chronic nephritis, alcoholism, and pulmonary tuberculosis.

PARASITES IN THE BLOOD

The following parasites have been detected in the blood: *Filaria sanguinis hominis*, hematozoan of malaria, protozoan of kala-azar, spirillum of relapsing fever, pneumococcus, micrococcus of Malta fever, bacillus of anthrax, typhoid fever, tetanus, tuberculosis, influenza, leprosy, glanders, bubonic plague, malignant edema, and diphtheria; the streptococcus, staphylococcus, meningococcus, gonococcus, trypanosoma, and colon bacillus.

OLIGOCHROMEMIA

Oligochromemia, or deficiency of hemoglobin, is usually proportionate to the reduction in the number of red cells; but there are two exceptions, namely, in chlorosis, in which disease the red cells may be reduced only 20 or 30 per cent., while the hemoglobin may be reduced 50 or 60 per cent., and in pernicious anemia, in which disease the blood-count is very low, while the corpuscles are relatively rich in hemoglobin.

The *color-index* represents the relation between the number of cells and the quantity of hemoglobin. In a patient having 2,500,000 red cells per cubic millimeter (50 per cent.) and 40 per cent. of hemoglobin, the color-index would be $\frac{40}{50} = 0.8$.

OLIGOCYTHEMIA

Oligocythemia, a diminution in the number of red cells, occurs in all forms of anemia, but it is especially intense after profuse or persistent hemorrhage, in pernicious anemia, in some cases of carcinoma, in chronic streptococcus infection, in acquired hemolytic jaundice, and in certain forms of intestinal helminthiasis (hookworm and fish tapeworm).

ANEMIA

Definition.—A deficiency in corpuscular substance, *i.e.*, a deficiency in red corpuscles, in hemoglobin, or in both, with or without changes in the total volume of the blood (Cabot).

Varieties.—(1) Secondary anemia; (2) primary anemia.

Symptoms.—Any form of anemia may present the following symptoms: Pallor of the skin and mucous membranes, loss of strength, and, in severe cases, febrile paroxysms.

Circulation.—A full, rapid pulse, unnatural pulsation of the cervical vessels, palpitation of the heart, a hemic murmur, a hum over the jugular vein, and slight dropsy, beginning in the

feet. In severe forms there may be ecchymoses and bleeding from mucous membranes.

Respiration.—Hurried breathing.

Digestion.—Dyspepsia.

Nervous System.—Headache, vertigo, disturbed sleep, neuralgic pains, and tendency to syncope.

SECONDARY ANEMIA

Definition.—A secondary anemia is one that is symptomatic of some conspicuous underlying condition.

Etiology.—Secondary anemia usually results from one of three causes: (1) Insufficient nutriment entering the circulation (inadequate food, chronic gastritis, cancer of the pylorus, etc.). (2) Excessive demands upon the blood-making organs (overwork, hemorrhage, chronic diarrhea, etc.). (3) Action of parasites or toxic agents (malaria, lead, syphilis, uremia, etc.).

Symptoms.—In addition to the ordinary phenomena of anemia the blood-count reveals a decrease in the number of red cells and a proportionate deficiency in the percentage of hemoglobin. The number of polymorphonuclear leukocytes is often increased. In severe form, microcytes, macrocytes, and poikilocytes are present, and occasionally nucleated red cells, chiefly normoblasts.

Prognosis.—This depends on the cause.

Treatment.—This includes the removal of the cause, if possible; the adoption of hygienic measures; and the use of iron, arsenic, and general tonics.

PRIMARY ANEMIA

Definition.—A primary anemia is one that, in the present state of our knowledge, cannot be associated with any conspicuous underlying cause.

Varieties.—Pernicious anemia and chlorosis. Splenic anemia is also regarded as primary anemia by some authorities.

PERNICIOUS ANEMIA

(Progressive Pernicious Anemia)

Definition.—A progressive form of anemia, of unknown origin, probably always fatal, characterized by intense oligocythemia, an embryonal type of blood formation, leukopenia, and gastric achylia.

Etiology.—As a rule no adequate cause is apparent. The disease usually appears about middle life, and is somewhat more frequent in males than in females. Forms of anemia closely resembling pernicious anemia may result from the action of intestinal parasites, especially the fish tapeworm (*Dibothriocephalus latus*) and the hookworm. The most plausible theory is that the disease is due to the hemolytic action of some poison produced within the body or the result of infection. According to W. Hunter, oral or gastric sepsis is an important factor.

Pathology.—The skin has a lemon-yellow hue, the subcutaneous fat is often well preserved, and the muscles are unusually red. The organs are pigmented and fatty. Iron pigment is especially abundant in the outer zones of the hepatic lobules. Marked atrophy of the gastric mucosa is sometimes observed. The bone-marrow, as a rule, is dark red, soft, and contains large numbers of nucleated red cells, especially macroblasts (probably as the result of an attempt to compensate for the excessive hemolysis). The hemolymph glands are frequently enlarged, congested, and pigmented. The spleen is sometimes enlarged. In the majority of cases there is found degeneration of the spinal cord, involving the posterior columns and to some extent the pyramidal tracts.

Symptoms.—The general symptoms are intense anemia, with its usual manifestations; a lemon-yellow tint to the skin; progressive weakness, without marked emaciation; attacks of moderate fever lasting a week or two; various gastro-intestinal disturbances, with paroxysms of epigastric pain; absence of free acid from the gastric juice; numbness, tingling, or other

paresthesias in the extremities; and not rarely hemorrhages, especially into the skin or retina; slight or moderate enlargement of the liver and spleen, and symptoms referable to the spinal cord, most often those of postero-lateral sclerosis.

The Blood.—The drop is pale and watery. There is a great reduction in the number of red cells, often to 1,000,000 or less; the hemoglobin is also reduced, but not proportionately (high color-index). The red cells usually show decided changes both in size (anisocytosis) and in shape (poikilocytosis). Nucleated red cells are more or less abundant. As a rule, the large forms (macroblasts) predominate. The leukocytes are usually decreased, though the lymphocytes are relatively increased.

Diagnosis.—*Parasitic anemia* may be recognized by the occurrence of eosinophilia and the discovery of the parasites or their ova in the stools.

Cancer rarely produces such extreme oligocythemia, the color-index is not high, macroblasts are rarely present, and there is often leukocytosis.

Aplastic Anemia.—This rapidly fatal form of pernicious anemia especially affects young women. In contrast with the ordinary form of pernicious anemia the course of the disease is acute, there is much greater tendency to hemorrhages, the color index is low, nucleated red cells are usually absent, and the percentage of lymphocytes is relatively very high (75 to 90 per cent.). Anatomically, the bone-marrow is yellow instead of red, the erythroblastic tissue being replaced by fat.

Prognosis.—Pernicious anemia usually ends fatally in from 2 to 5 years. It is doubtful whether recovery ever occurs. Remissions lasting from a few weeks to several years occur in the majority of cases.

Treatment.—Fresh air, rest, and a diet as liberal as the digestive power of the patient will permit are requisite. Warm salt baths and massage are valuable adjuvants to internal treatment. The teeth should receive careful attention. If there is pyorrhea, this should be carefully treated.

Arsenic is the most valuable drug. It may be given in the form of Fowler's solution, the dose being gradually increased from 2 or 3 to 15 or 20 minims three times a day. Iron is rarely of service. Inhalations of oxygen have been recommended (Shattuck). Transfusion of fresh unmodified blood (300 to 800 c.c. at intervals of from a week to month) is often more effective than any other measure. Digestive disturbances are often benefited by the administration of diluted hydrochloric acid and a bitter.

CHLOROSIS

(Green Sickness)

Definition.—A form of anemia occurring exclusively in young women and characterized by marked oligochromemia.

Etiology.—The disease usually occurs between the fifteenth and twenty-fifth years. Heredity, poor hygienic surroundings, and overwork are predisposing factors. The real cause of the disease has not been determined.

Pathology.—In some fatal cases imperfect development of the vascular and generative systems has been observed.

Symptoms.—In addition to the general symptoms of anemia the conspicuous features are a greenish hue of the skin; pallor and weakness without marked loss of flesh; dyspepsia with perversion of appetite; menstrual disorders, especially amenorrhea; and a tendency to hysteric outbreaks. The *blood changes* are characteristic. The number of red cells is moderately reduced (not often below 3,500,000); the hemoglobin, on the other hand, is greatly reduced—usually to below 50 per cent. There is no leukocytosis.

Complications.—Complications are uncommon, but peptic ulcer, hyperthyroidism, tuberculosis, or thrombosis of the cerebral sinuses or veins of the extremities may occur.

Prognosis.—The prognosis is good, but relapses are common.

Treatment.—Fresh air, sunlight, open-air exercise, and nourishing food are valuable aids in treatment. Very severe

cases require complete rest in bed. If there be a good reaction, warm baths, followed by short cold douches, are efficacious. Iron is almost a specific. It is most frequently prescribed in the form of Blaud's pills, of which the dose is three pills, gradually increased to nine, a day.

Laxatives, preferably mild salines, rank next in importance to iron. Arsenic is distinctly less valuable than iron. Superacidity of the gastric juice is best treated by alkalis.

LEUKEMIA

(Leukocythemia)

Definition.—A disease characterized by an overgrowth of the leukoblastic tissues and an enormous increase in the number of circulating leukocytes.

Etiology.—The causes are obscure. More males are affected than females. The disease occurs most frequently in middle life. Heredity is without influence. Repeated exposures to x -rays or to radium seem to have been a factor in a few instances. An infectious origin has been suggested.

Varieties.—The bone-marrow, spleen, and lymph glands are all more or less involved in the hyperplasia, but according as the latter is of the myelocytic or of the lymphocytic type two forms of the disease are recognized: (1) myeloid (splenomedullary) leukemia; (2) lymphoid leukemia. Either form may be acute or chronic.

Pathology.—In the *myeloid form* the fat of the marrow, especially of the long bones, is largely replaced by a grayish-white or grayish-red tissue, which microscopically exhibits numerous myelocytes and closely related cells. The spleen and liver are much enlarged, the former from myeloid transformation (metaplasia), the latter from packing of the capillaries with leukocytes similar to those in the marrow and spleen. Some of the lymph glands also show evidences of myeloid transformation.

In the *lymphoid form*, although enlargement of the lymph gland is usually the most striking feature, the changes are not

limited to these parts of the hemopoietic system. The marrow is always affected and not rarely the spleen also shares in the process. The cellular transformation and proliferation, however, instead of being myelocytic, are lymphocytic, large or small lymphocytes preponderating according as the disease is acute or chronic.

Symptoms.—*Myeloid Leukemia.*—Progressive loss of flesh and strength and an increase in the size of the abdomen due to the enormous enlargement of the spleen are early manifestations. Pain in the splenic region is often experienced. The liver is moderately enlarged. Hemorrhages from the mucous membranes and into the skin, retina, or brain are common. Febrile attacks, usually lasting a week or two, occur in almost every case. The urine contains an excess of uric acid. Anemia is not an early symptom, but it always supervenes as the disease advances, and in the later stages pallor, dyspnea, digestive disturbances, edema, etc., may be conspicuous features. Hemorrhage or leukemic infiltration may cause dimness of vision if it occurs in the retina or optic nerve; deafness if it occurs in the labyrinth; or exophthalmos if it occurs in the orbit. Priapism is an occasional symptom.

The blood changes are characteristic. The number of leukocytes is enormously increased, the count usually ranging between 100,000 and 500,000. The increase is made up of all the normal cells and a large number, usually between 30 and 60 per cent., of myelocytes. The eosinophiles and mast cells are both relatively and absolutely increased. In advanced cases the number of red cells and the percentage of hemoglobin are considerably decreased.

Lymphatic leukemia is much less common. The general symptoms are much the same as in myeloid leukemia. The most conspicuous feature, however, is enlargement of the lymph glands—cervical, axillary, inguinal, etc. The spleen is more or less enlarged in almost every case. Enlargement of the tonsils is sometimes an early symptom suggesting tonsillitis. The striking change in the blood is the marked increase in the

lymphocytes. The total leukocyte count usually ranges between 50,000 and 200,000, and the increase consists almost exclusively of lymphocytes.

Acute Leukemia.—This form is observed chiefly in children. Ulcerative or pseudomembranous inflammation of the mouth or throat often marks the onset. Hemorrhages are common. Pallor, weakness, emaciation, and an irregular fever soon supervene. The lymph-nodes and spleen are usually, but not invariably, enlarged. The leukocyte count is increased, small or large mononuclear forms predominating according as the disease is of the *lymphoid* or *myeloid* type. The course is progressively downward.

Atypical Leukemia.—Cases of leukemia are rarely observed in which the leukocytes of the blood are approximately normal, but there is an absolute and often a relative increase of certain mature or immature white cells (aleukemic leukemia). Occasionally, also, cases occur in which the blood-picture suggests a combination of pernicious anemia and myeloid leukemia (leukanemia). Again, there is an atypical form of acute or subacute leukemia (chloroma), in which green-tinted tumor-like masses appear in certain bones, especially those of the skull and invade the surrounding tissues.

Diagnosis.—The ordinary chronic cases present no diagnostic difficulties, but atypical forms may go unrecognized and acute cases may readily be mistaken for certain infectious diseases, especially typhoid fever and septicemia, unless the blood be carefully studied. It must not be forgotten that under the influence of infections the leukocyte count may become temporarily almost normal.

Prognosis.—The disease is always fatal. The average duration of the chronic forms is from two to three years. Remissions are not uncommon. Acute cases last from a few weeks to several months. Death may result from exhaustion or an intercurrent infection, such as tuberculosis, pneumonia, or septicopyemia.

Treatment.—An effort should be made to maintain the general nutrition by regulating the diet and attending to hygienic measures. Rest is often advisable. Among drugs, benzol appears to possess some potency, although it is not free from dangerous properties. From 10 to 20 drops, with equal amounts of olive oil, may be given in an emulsion three or four times a day. Blood-counts should be made at frequent intervals and the treatment should be suspended when the number of leukocytes has fallen to 30,000. Arsenic is also serviceable. The use of the x-rays is often followed by marked, though temporary, improvement. Operative treatment is of no avail.

HODGKIN'S DISEASE

(Pseudoleukemia)

Definition.—A comparatively rare disease characterized by enlargement of one or more groups of lymph-glands and progressive anemia.

Etiology.—The causes are unknown. It is most commonly seen in young adults of the male sex. Tuberculosis is sometimes associated with it, but the view that the disease is a form of tuberculosis has been abandoned. The reported finding of spirochetes has not been confirmed.

Pathology.—There is marked enlargement of the lymph-glands—cervical, axillary, inguinal, thoracic, retroperitoneal, etc. Microscopically, the changes consist of hyperplasia of the lymphoid cells with proliferation of the endothelial and reticular cells, formation of characteristic giant cells with several indented nuclei (Dorothy Reed cells), and progressive fibrosis. It is doubtful whether a sharp line of demarcation can always be drawn between the lymphomas of Hodgkin's disease and malignant lymphomas (lymphosarcomas) or even some leukemic lymphomas. Nodules of lymphoid tissue are often found in the spleen, liver, bone-marrow, etc.

Symptoms.—The symptoms resemble those of lymphatic leukemia, but the blood changes are not characteristic. There

is pronounced secondary anemia, with little if any increase in the number of leukocytes. The swollen lymph nodes are smooth, of varying consistency, painless, and almost never confluent. Symptoms, the result of local pressure—dyspnea, cough, cyanosis, edema, neuralgic pains, etc.—are often present. The spleen and liver are frequently enlarged. Periods of fever occur in almost every case. The disease is invariably fatal and the duration varies from a few months to several years.

Diagnosis.—*Tuberculous adenitis* may usually be distinguished by the tendency of the enlarged glands to fuse, to become adherent to the skin, and to suppurate and discharge. In doubtful cases the diagnosis must rest upon the microscopic examination of one of the enlarged glands excised for the purpose.

In *sarcoma* the growths develop, as a rule, more rapidly, are often painful, and show a marked tendency to fuse, to involve their capsules, and to invade adjacent structures. In some instances, however, the differential diagnosis may not be possible without a microscopic examination of an excised gland.

Treatment.—The treatment is that of leukemia.

HEMOPHILIA

Definition.—An hereditary constitutional defect characterized by immoderate and persistent hemorrhages after the most trivial injuries.

Etiology and Pathogenesis.—Nothing is known of the causes of the disease beyond the facts that it is strongly hereditary and familial, that it occurs chiefly in males, and that it is transmitted almost exclusively by unaffected females. The blood of hemophilic patients has a prolonged coagulation time; according to Sahli, this is due to a deficiency of thrombokinase produced in the walls of the blood-vessels and other tissues, but according to Howell it depends upon a decrease of prothrombin derived from the blood platelets.

Symptoms.—The chief symptom is the occurrence of profuse, at times uncontrollable, hemorrhages, usually following slight trauma, but not rarely developing spontaneously. Bleedings from the mucous membranes and into the subcutaneous tissues are very common, and hemarthrosis, sometimes resulting in inflammatory changes in the joints and suggesting rheumatic or tuberculous arthritis, is also frequent.

Prognosis.—The outlook is unfavorable in most cases. About 60 per cent. of all “bleeders” die before the eighth year, and only about 11 per cent. reach maturity. In some instances the tendency is outgrown.

Treatment.—This is chiefly protective and palliative. The most serviceable remedies are blood-serum and fresh unmodified blood. The serum may be given subcutaneously or intravenously (30 mls daily). In severe cases blood transfusion (100–500 c.c.) is preferable. Calcium lactate is of doubtful value. Thyroid extract has proved efficacious in some instances. Local hemorrhages should be treated by rest, compression, and the application of hemostatics (fresh or dried blood-serum, adrenalin, thromboplastin, coagulen, etc.). Hemophilic joints should be treated by rest and moderate compression and after a few days subjected to gentle massage.

PURPURA

Purpura, or the occurrence of spontaneous hemorrhages into the skin and mucous membranes, is always a *secondary* condition, that is, a symptom of some underlying morbid state. Since, however, the cause of several purpuras is not known, it is customary to speak of a *primary* or *essential* type.

SECONDARY PURPURA

Secondary purpura occurs in the course of the following conditions: (1) *Acute infectious diseases*. A purpuric eruption is usually present in typhus fever and may occur also in cerebrospinal fever, septicopyemia (ulcerative endocarditis), measles, smallpox, etc. (2) *Cachectic states*, such as develop

in carcinoma, scurvy, pernicious anemia, leukemia, chronic nephritis, old age, etc. (3) *Certain intoxications*, notably poisoning by iodids, antipyrin, salicylic acid, quinin, and mercury. Apart from drug poisoning, snake bite and jaundice may also cause purpura. (4) *Venous stasis*. Purpura from this cause may occur about tight bandages or after epileptic seizures or the paroxysms of whooping-cough. (5) *Nervous disorders*. The purpuras occasionally observed in hysteria and rarely in association with neuralgia, sciatica, and the lightning pains of tabes dorsalis belong in this group.

PRIMARY PURPURA

Etiology and Pathogenesis.—The cause of primary purpura is unknown. The disease occurs most frequently in the second and third decades and is more common in males than in females. The view that it is caused by the action of some unknown toxin on the capillaries or on the blood itself has many advocates. The number of blood platelets is often greatly diminished; the blood coagulates within the normal time, but the clot contracts slowly and imperfectly.

Symptoms.—Several varieties are recognized, but the barrier separating them is not very sharply defined.

Purpura Simplex.—In this form the chief feature is the appearance in successive crops of small hemorrhagic spots on the extremities, especially the legs. Pains in the joints are frequently noted, and when these are pronounced the condition is usually designated *purpura rheumatica* (Peliosis rheumatica of Schönlein). Urticaria is also common. The duration is from a few days to several months.

Henoch's Purpura.—This form is characterized by recurring outbreaks of purpura and attacks of abdominal pain, often accompanied by vomiting and diarrhea (exudative erythema of the bowel). The discharges are not rarely bloody. Arthritis is common. Erythema, urticaria, and localized edema are noted in many cases. Nephritis is a frequent com-

plication. The condition occurs most often in early life and seems to be closely related to angioneurotic edema. The duration of the entire illness is from two or three weeks to as many months.

Purpura Hemorrhagica (Morbus Maculosus of Werlhof).—In this form there is bleeding from the mucous membranes, especially the nose, mouth, and genito-urinary tract, as well as into the skin. Fever is present in more than half of the cases. Arthritis and nephritis are not infrequent. Anemia soon develops in severe cases. The average duration is from four to six weeks. Relapses are common. In rare instances the disease assumes a malignant character (*purpura fulminans*) and ends fatally within a week or two; on the other hand, it occasionally becomes chronic and lasts for many years.

Associations.—Urticaria, erythema multiforme, erythema nodosum, localized edema (angioneurotic edema), and nephritis are of frequent occurrence in all forms of primary purpura. Nephritis is especially common in Henoch's purpura and may prove fatal. Endocarditis, pericarditis and cerebral hemorrhage are rare complications.

Diagnosis.—*Scurvy* may be distinguished by a history of dietetic errors, by the spongy state of the gums, and by the brawny induration of the muscles. In *hemophilia* the hemorrhagic tendency is hereditary and congenital. *Secondary purpura* may usually be differentiated by the history and the other symptoms of the primary condition. Care must be taken not to mistake the abdominal crises of Henoch's purpura for intussusception or appendicitis.

Prognosis.—Recovery is the rule, except in the severe forms. In fatal cases death may be caused by cerebral hemorrhage, nephritis, or exhaustion.

Treatment.—The patient should be put to bed and given light nourishing food. The most useful remedies are transfusion of whole blood, subcutaneous injections (30 c.c. daily) of fresh blood serum, and subcutaneous injections of an extract of blood-platelets (coagulen: 5 grams in 300 c.c. of saline solu-

tion). Oil of turpentine and calcium lactate have been recommended. Iron is useful during convalescence.

ERYTHREMIA

(Vaquez's Disease)

Erythremia is a rare affection characterized by persistent redness or cyanosis of the skin, polycythemia (7 to 13 millions per c.mm.), and marked enlargement of the spleen. Less common symptoms are high blood-pressure, nervous symptoms (headache, vertigo, neuralgic pains, and ready fatigue), and clubbing of the fingers. Hemorrhage is not infrequent, especially cerebral hemorrhage. As a rule, the disease lasts many years, a fatal event ultimately occurring from heart failure, hemorrhage or peripheral venous thrombosis. Hyperactivity of the erythroblastic tissues of the bone-marrow is believed to be the basis of the disorder. In making the diagnosis one must exclude other causes of persistent cyanosis, such as chronic cardiac and pulmonary disease, drug poisoning (acetanilid, antipyrin, and other coal-tar products), the rare forms of chronic enteritis in which cyanosis occurs probably from the absorption of nitrites or hydrogen sulphide from the intestines (enterogenous cyanosis), and massive tuberculosis of the spleen. Benefit has resulted in some cases from x-ray or radium treatment of the bones, venesection, and hydrotherapy.

DISEASES OF THE DUCTLESS GLANDS

ADDISON'S DISEASE

Definition.—A rare disease due to inadequacy of the adrenal glands or other parts of the chromaffin system and characterized by extreme muscular and circulatory weakness, pigmentation of the skin and mucous membranes, and gastrointestinal irritability.

Etiology.—The disease occurs most frequently between the ages of twenty and fifty years, and is more common in males than in females. The causes of tuberculosis in general favor its development.

Pathology.—The usual lesion is tuberculosis (fibrocaceous) of the adrenals. Occasionally there is simple atrophy, fibrosis, or tumor. In some instances degenerative changes are found in the adjacent sympathetic trunks or ganglia either in connection with, or in absence of, changes in the adrenals.

Symptoms.—Profound muscular weakness, developing without obvious cause and unaccompanied by any commensurate emaciation, is an early symptom. The arterial pressure is usually very low and in advanced cases attacks of syncope are of frequent occurrence. Brownish pigmentation or bronzing of the skin, almost universal, but especially marked on the exposed parts and genitalia, around the nipples and navel, and wherever the skin has been irritated or compressed, appears sooner or later in nearly every case. In many cases the mucous membranes are also affected. Gastro-intestinal irritability, manifested by anorexia, epigastric discomfort or

pain, and attacks of vomiting or diarrhea, is also common. The blood shows the usual changes of secondary anemia.

Diagnosis.—This is difficult until the symptoms are well developed. Other forms of pigmentation, such as that occurring in chronic jaundice, malarial melanemia, melanotic sarcoma, hemochromatosis (bronzed diabetes), pregnancy, malignant disease within the abdomen, pellagra, argyria, chronic arsenic poisoning and vagabond's disease (discoloration resulting from uncleanly habits and exposure), etc., must be excluded.

Prognosis.—The disease is probably always fatal, and usually lasts from a few months to several years. The course is not uniform but marked by remissions and exacerbations. Death is usually due to asthenia, but it may occur from syncope or a toxic condition (convulsions, delirium, coma).

Treatment.—The general treatment includes rest, a nutritious but easily digestible diet, and the administration of tonics. In some cases temporary good effects have followed the use of the extract of the suprarenal gland in doses of from 15 to 20 grains thrice daily.

EXOPHTHALMIC GOITER

(Hyperthyroidism; Graves' Disease; Parry's Disease; Basedow's Disease)

Definition.—A disease characterized by enlargement of the thyroid gland, marked prominence of the eyeballs, tachycardia, muscular tremors, and increased metabolism.

Etiology.—The disease most frequently develops in the third or fourth decade, and affects women much more often than men. In many instances it is consequent upon acute disease, pregnancy, the menopause, or profound mental or emotional strain.

Pathology.—The pathogenesis of Graves' disease is still undetermined. Excessive functional activity of the thyroid gland (hyperthyroidism) is undoubtedly the basis of the disorder, but the initiating cause is unknown. The essential

changes in the gland are diffuse proliferation of the epithelium and diminution of the colloidal material.

Symptoms.—*Cardiac Phenomena.*—Acceleration of the pulse (100 to 150) and palpitation are constant features. Both are intensified by excitement. Hypertrophy of the heart may ultimately ensue from overaction or thyroid intoxication. A soft systolic murmur is frequently heard at the apex.

Ocular Phenomena.—Exophthalmos, or protrusion of the eyeballs, usually bilateral, is an obtrusive symptom. Accompanying it there are often other ocular changes, such as lagging of the upper eyelids in downward movement of the eyeballs (von Graefe's sign); a peculiar staring look, due to widening of the palpebral fissure (Dalrymple's sign); infrequent and incomplete reflex winking (Stellwag's sign); insufficient power of convergence for near objects (Möbius' sign). As a rule, vision is not disturbed.

Thyroid Phenomena.—Enlargement of the thyroid may be the last symptom to appear. One or both lobes of the gland may be affected. Palpation often detects pulsation and a purring thrill, and auscultation, a soft systolic bruit.

Nervous Phenomena.—A fine muscular tremor, most pronounced in the extremities, is an early symptom. Abnormal irritability and extreme restlessness are characteristic of the disease. Vasomotor disturbances, such as excessive flushing and sweating, urticaria, and local edema, are frequently observed. Mania or melancholia occasionally supervenes.

General Symptoms.—As the disease progresses, weakness, emaciation, and anemia usually become pronounced. Attacks of vomiting and of serous diarrhea are common. Moderate fever is an occasional symptom. There may be glycosuria and albuminuria.

Increased Metabolism.—The basal metabolic rate is increased from +15 to +50 or even +75. This factor is responsible for the weakness and emaciation.

Diagnosis.—Only the incomplete forms (*formes frustes*) are likely to escape recognition. Tachycardia is never absent, but neither goiter nor exophthalmos is an essential symptom.

Prognosis.—The disease usually runs a protracted course, extending over many years. Some patients (probably 30 per cent.) completely recover, many improve, and a few die within a few weeks or a few months (acute form). Remissions and relapses are common. Occasionally myxedema supervenes. Death may result from heart failure (sudden or gradual), intercurrent disease, or exhaustion.

Treatment.—The general nutrition should be improved by rest, freedom from excitement, a generous, readily digestible diet, and hydrotherapy. In severe cases absolute rest in bed is an essential point in the treatment. Applications of cold, by means of Leiter's tubes or ice-bags, to the precordium lessen the palpitation.

Belladonna is undoubtedly of value in many cases. It should be given in ascending doses until some dryness of the throat is produced. If the circulation is feeble, digitalis may be found of service; on the other hand, if the heart is strong, better results may be obtained with aconite or veratrum viride. If anemia exists, iron and arsenic are useful. Bromids are of service in controlling nervous symptoms. Starr has observed improvement from the use of sodium glycerophosphate in doses of 20 grains three or four times a day. The consensus of opinion is adverse to the use of thyroid extract.

The milk and serum of thyroidectomized animals have proved disappointing. Applications of *x*-rays to the thyroid are sometimes of service. When medical measures fail after a fair trial operative treatment (partial thyroidectomy, ligation of one or more of the thyroid arteries) is indicated, but it should not be undertaken during acute exacerbations. After operation medical supervision is always necessary.

SECONDARY HYPERTHYROIDISM

(Toxic Goiter)

These terms are applied to a symptom-complex supervening after an interval of years upon ordinary goiter. The condition

usually develops about middle life, and emotional stress, overwork, or focal infection may be an exciting factor. The change in the thyroid is, as a rule, of an adenomatous nature. The *symptoms* resemble those of exophthalmic goiter, but exophthalmos is rare, thrill and bruit over the thyroid are uncommon, arrhythmia and other indications of myocardial involvement develop earlier, and so-called crises are exceptional. The *treatment* is in the main that of exophthalmic goiter.

MYXEDEMA; HYPOTHYROIDISM

Definition.—A chronic disease due to a lack of thyroid secretion and characterized by brawny thickening of the subcutaneous tissues, loss of energy and mental deterioration.

Etiology.—The disease is much more frequent in women than in men. It is occasionally hereditary or familial. It usually develops between the ages of twenty and fifty years. The basic condition is atrophy of the thyroid gland, but the cause of this morbid change is unknown.

A congenital form of myxedema is observed in *cretinism*, and an analogous condition (*operative myxedema* or *cachexia strumipriva*) frequently follows total extirpation of the thyroid gland.

Symptoms.—It is manifested by a gradual swelling of the subcutaneous tissues, particularly of the face, supraclavicular regions, and hands. Unlike edema, the parts do not pit on pressure, but are firm and elastic. The skin is dry and harsh. The hair becomes brittle and falls out. The thyroid gland is atrophied. A peculiar slowness in thought, speech, and movements is a characteristic symptom. Eventually dementia may supervene. The temperature of the body is subnormal and the pulse is infrequent. There is impairment of the special senses. Sensory phenomena, such as undue sensitiveness to cold, neuralgic pains, and formication are common. Albuminuria and glycosuria sometimes occur. The sexual functions are depressed.

The rate of basal metabolism is always reduced, ranging from -15 to -40 , according to the severity of the case.*

In *cretinism*, which may be endemic or sporadic, there is arrested development, physical and mental, with changes in the skin like those in myxedema and a characteristic deformity of the bones and soft parts. The head is large, the features are coarse and bloated, the expression is stolid or idiotic, the trunk and limbs are short and thick, the abdomen is protuberant, the sexual organs are infantile, and the skin is rough and dry.

The symptoms of *cachexia strumipriva* are much the same as those of spontaneous myxedema in the adult.

Diagnosis.—Mild cases are likely to be overlooked unless examined very carefully. *Chronic nephritis* may give rise to confusion, but in this disease the swelling pits on pressure, the skin is not thickened and indurated, the hair does not fall, and there is no peculiar slowing of the mental processes. In *acromegaly* the face is elongated, the chin is prominent, kyphosis is common, the hair grows well, and the hypertrophy affects the bones as well as the soft parts.

Cretinism is sometimes confused with mongolian idiocy and with achondroplasia. In the *mongolian idiot*, however, the eyes are obliquely set, the subcutaneous tissues are not thickened, and there is restlessness and vivacity rather than inertness and stolidity. In *achondroplasia* the limbs are very short, with the hands and feet attached almost directly to the trunk, the intellect is good, and the skin is natural.

Prognosis.—Marked improvement or even symptomatic cure is possible provided appropriate treatment is begun early and continued throughout life. In the absence of specific therapy the tendency of the disease is to become progressively worse.

Treatment.—As patients with myxedema are extremely susceptible to low temperatures, they should be warmly clad and protected from exposure to cold. Residence during the

* Normally, the rate ranges between -15 and $+15$.

winter in a warm, sunny climate is desirable. Warm baths are often beneficial. Modern treatment consists in the administration of extract of sheep's thyroid (from 1 to 5 grains thrice daily, the final dose depending upon individual need or tolerance).

ACROMEGALY

Definition.—A chronic disease depending upon changes in the pituitary body and characterized by abnormal growth, especially of the hands, feet, and face.

Etiology.—The cause is unknown. The disease is more common in males than in females, and usually begins between the twentieth and fortieth years.

Pathology.—In the vast majority of cases the pituitary body is the seat of hyperplasia or tumor formation (benign or malignant). Other ductless glands, especially the thyroid and the thymus, may also be enlarged. Hypertrophic changes are observed in the bones and subcutaneous tissues and in some cases also in the heart, liver, and spleen. The symptoms are probably due to hyperactivity of the pituitary body, especially of the anterior lobe (hyperpituitarism).

Symptoms.—Gradual enlargement of the hands, feet, and face is the most conspicuous symptom. The face is elongated, the nose large and bulbous, the lower jaw massive and prominent (prognathism), and the lips and tongue are thick. The hands are large and spade-like, and the fingers sausage-shaped. The feet are affected in a similar way. Kyphosis is almost invariably present. Impotence in the male and amenorrhea in the female are early manifestations. Symptoms of gross intracerebral disease, especially headache and defect in sight (hemianopsia), often appear. Muscular weakness, mental hebetude, excessive thirst, and glycosuria are frequently noted in advanced cases. Cardiovascular disease, diabetes, myxedema, and exophthalmic goiter are not uncommon associations. The disease is progressive but usually

lasts many years. If the lesion of the hypophysis is malignant, however, the duration may be only two or three years.

Diagnosis.—This is rarely difficult except in the earliest stages. In *Paget's osteitis deformans* the long bones are especially involved and are not only enlarged but are considerably deformed, and the head is broad at the top. The distinctive features of *myxedema* have already been stated (see p. 184).

Treatment.—Medical treatment is unsatisfactory. Thyroid extract and thymus extract have been used with apparent benefit in a few cases. Removal of the pituitary tumor has been successfully accomplished by Cushing and others in a number of instances.

HYPOPITUITARISM

The condition first described by Frölich under the name of **dystrophia adiposo-genitalis** and characterized by general obesity, hypoplasia or atrophy of the sexual organs, a lack of pubic hair, somnolence, and a high tolerance to carbohydrates is apparently due to insufficiency of the hypophysis, although it is not improbable that other glands of internal secretion may also be concerned in the process. Closely related conditions are:

(1) **Adiposis Dolorosa (Dercum's Disease).**—In this disorder there are more or less symmetrical deposits of fatty masses in various parts of the body, with tenderness of the affected areas, neuritic or neuralgia pains elsewhere, varying degrees of asthenia, and, not rarely, more or less psychic disturbance.

(2) **Ateliosis.**—The chief features of this condition, which is nearly the antithesis of acromegaly, are a childish facial appearance, diminutive stature, with short slender limbs, ill-developed muscular prominences, small jaw bones, a thin piping voice, low arterial tension, and scanty urination.

(3) **Idiopathic Infantilism (Lorain Type).**—In this disorder the stature is diminutive and there is imperfect development of the sexual organs and the secondary sexual characteristics, but the outlines of the body are those of a well-formed adult.

(4) **Progeria.**—This is a form of infantilism associated with premature senility and sclerotic changes in the cardiovascular system.

TETANY

Definition.—A rare disease characterized by bilateral, intermittent, tonic spasms, especially of the extremities, and increased excitability of the nerves and muscles to mechanical and electrical stimulation.

Etiology.—Postoperative and non-operative cases are observed. Postoperative tetany sometimes follows operations upon the thyroid (removal or destruction of the parathyroids). Non-operative tetany in adults is most commonly seen as a complication of gastrectasis, but it may occur in other severe gastro-intestinal affections, in pregnancy or with lactation, in acute infectious diseases, or in poisoning from certain substances, such as alkalies, ergot, etc. In Austria it is sometimes endemic, chiefly among handworkers—shoemakers, tailors, etc. In young children it is usually associated with rickets. The researches of MacCallum and Voegtlin indicate that tetany is due to a lack of calcium in the blood, the result of parathyroid insufficiency.

Symptoms.—The patient is seized with bilateral tonic spasms, beginning in the hands and feet and spreading upward. In many instances only the hands are affected. The muscles of the trunk and face are seldom involved. The spasms usually occur in paroxysms lasting from a few minutes to an hour or two, but in very severe cases they may be more or less continuous. They are sometimes attended by pain. Laryngismus stridulus is not uncommon in children. The mind is usually unaffected. Occasionally the cramps are accompanied by slight fever and edema. Even when the contractions have ceased they may be reproduced by the application of pressure over the nerve trunks or vessels of the affected member (*Trousseau's sign*). Sometimes a mere tap over the nerve is sufficient to excite spasm (*Chvostek's sign*). The electrical excitability of the motor nerves is also increased (*Erb's sign*). Percussion or electrical stimulation of sensory nerves not infrequently calls forth abnormally intense sensa-

tions (*Hoffmann's sign*). Very mild attacks (*latent tetany*) may consist of paresthesia, muscular irritability, and slight stiffness without spasm.

Diagnosis.—In *tetanus* a source of infection is usually found; trismus or lockjaw appears early, and the muscles of the back are more contracted than those of the limbs.

Hysteria may be distinguished from tetany by the history, the emotional disturbances, the unilateral character of the spasms, and the absence of Trousseau's sign.

Prognosis.—The attacks may occur at intervals of hours or days, and the entire disease may last from a few days to several months. Relapses are common. The outlook, generally speaking, is good, although the mortality is high in tetany arising in gastrectasis.

Treatment.—The cause should be sought for and removed if possible. When there is rickets cod-liver oil and phosphorus are of value. Calcium salts (15 grains of the lactate four times a day) and parathyroid preparations are the most serviceable remedies. In cases of emergency calcium lactate (500 c.c. of a 5 per cent. solution in normal saline solution) should be given intravenously with subcutaneous injections of emulsion of freshly prepared parathyroid gland. Bromids, chloral, or hyoscin may be used as adjuvants. Warm baths are beneficial. Surgical intervention is sometimes indicated in gastrectasis.

PRIMITIVE SPLENOMEGALY WITH ANEMIA

(Banti's Disease; Splenic Anemia)

This is a chronic disease of unknown origin, characterized by progressive enlargement of the spleen, anemia of the secondary type, leukopenia, a marked tendency to hemorrhages, and, in certain cases after the lapse of years, cirrhosis of the liver, with ascites and slight jaundice (Banti's disease).

The splenic enlargement is very pronounced and for many years may be the only conspicuous feature. The blood-picture

is, as a rule, that of moderate anemia (3,000,000 to 4,000,000 erythrocytes per c.mm.), with a low color-index and marked leukopenia. The hemorrhages are usually from the stomach or bowel and are often profuse. Cirrhosis of the liver, with enlargement or atrophy of the organ, ascites, jaundice, etc., is a late phenomenon, and the one to which Banti directed special attention. The pathologic changes in the spleen consist of an overgrowth of connective tissue, hyperplasia of the endothelial cells, and endophlebitis of the splenic vein. The portal vein may also be affected. The disease is remarkably chronic, often lasting ten years or longer. Done at the proper time, splenectomy frequently effects a complete cure. The mortality of the operation in 118 cases was 19 per cent.

OTHER CHRONIC ENLARGEMENTS OF THE SPLEEN

Chronic enlargement of the spleen other than that occurring in Banti's disease is observed in the following conditions:

(1) **Tumors and Cysts.**—Sarcoma, endothelioma, echinococcus cysts, etc. Many observers regard the Gaucher type of splenomegaly as a neoplasm. A characteristic feature of this disease is the presence of peculiar large cells (20–40 microns in diameter) in the spleen, lymph-glands, and bone-marrow. Clinically, it closely resembles Banti's disease, but is distinguished by its family occurrence and predilection for childhood, by the early appearance of a brownish-yellow discoloration (non-icteric) of the face and hands, and wedge-shaped thickening of the conjunctiva, and by the presence of ascites.

(2) **Diseases of the Blood-making Organs.**—Leukemia, pernicious anemia, erythemia, etc. The definite blood-changes in these conditions are characteristic.

(3) **Chronic Infections.**—Malaria, syphilis, tuberculosis, kala-azar, etc. Syphilis is probably the commonest cause of infantile splenomegaly. Massive tuberculosis of the spleen may closely resemble Banti's disease.

(4) **Disorders of Nutrition.**—Rickets, amyloid disease, certain obscure gastro-intestinal intoxications, etc.

(5) **Cirrhosis of the Liver.**—In the later stages the clinical picture may be like that of Banti's disease. The early appearance and marked prominence of the hepatic symptoms and, in Hanot's cirrhosis, the occurrence of leukocytosis are important diagnostic features.

(6) **Passive congestion of the spleen,** the result of chronic heart disease, primary pylephlebitis, and other obstructive disorders of the portal circulation.

(7) **Chronic Family Acholuric Jaundice** (Splenomegalic Jaundice).—This condition is described on p. 95.

(8) **Anemia in Infancy and Early Childhood.**—This includes a large ill-defined group of cases, some of which are little understood. In many cases the anemia and splenic enlargement are clearly secondary to such diseases as syphilis, rickets, scurvy, etc., but in others the cause is not obvious. In the latter group are included examples of Banti's disease or Gaucher's disease, acholuric jaundice, aberrant forms of leukemia, and the anemia pseudoleukemia infantium of von Jaksch.

DISEASES OF THE CIRCULATORY SYSTEM

GENERAL SYMPTOMATOLOGY

INSPECTION

INSPECTION serves to determine the position, force, and extent of the apex-beat and to detect any unnatural prominences in the region of the heart or aorta and any abnormal centers of pulsation.

The Apex-beat.—In healthy adults the apex-beat is in the fifth left intercostal space, about half an inch internal to the midclavicular line. In young children it is commonly in the fourth interspace, and in old persons it is often as low as the sixth interspace.

The Effect of Respiration and Position upon the Apex-beat.—The location and force of the apex-beat are modified by the posture of the patient and the stage of the respiratory act. In the recumbent position the apex-beat may be elevated half an inch or more, and when the body is inclined to the left, the heart being a more or less movable organ, the beat may be detected in the midclavicular line, or even some distance to its outer side.

During forced inspiration the beat may become imperceptible or, if such is not the case, it may be found some distance below its usual place, on account of the upward movement of the ribs in the inspiratory act. During forced expiration the beat becomes more forcible, and its position elevated on account of the descent of the ribs which occurs in expiration.

In view of the influence exerted by respiration and position upon the apex-beat, the patient, as a rule, should be examined in the erect or sitting posture, while breathing quietly.

Displacement of the Apex-beat.—*Displacement to the left* may result from:

1. Hypertrophy or dilatation of the heart (down and to the left).
2. Chronic diseases of the left lung and pleura, associated with retraction—as fibroid phthisis and pleural adhesions.
3. Abdominal tumors and effusions (up and to the left).
4. The pressure of a pleural effusion on the right side (up and to the left).

Displacement to the right may be caused by:

1. Chronic disease of the right lung or pleura associated with retraction.
2. Pressure of a pleural effusion on the left side.
3. Transposition of the viscera.

Displacement downward may result from:

1. Hypertrophy or dilatation of the heart, chiefly of the left ventricle.
2. Pressure of solid growths in the upper mediastinum.
3. Aneurysm of the aortic arch.

Deformity of the chest from spinal curvature may also cause considerable displacement of the heart.

Changes in Force and Extent of the Apex-beat.—*The force and extent of the pulsation may be increased* by:

1. Hypertrophy of the heart.
2. Forcible action of the heart caused by emotional or physical excitement, reflex irritation, drugs, Graves' disease, etc.
3. Thinning of the chest-walls and shrinking of the lungs, as in pulmonary tuberculosis.

The strength of the apex-beat may be lessened by:

1. Degeneration or dilatation of the heart.
2. Pericardial effusion.
3. Emphysema.
4. Collapse or shock

Abnormal Centers of Pulsation.—*Epigastric pulsation* may result from:

1. Excited action of the heart from any cause.
2. Enlargement of the right ventricle.
3. Dynamic throbbing of the aorta, noted in certain nervous and anemic patients.

4. Low position of the heart from arteriosclerosis.

5. Aneurysm of the abdominal aorta.

6. Tumors of the left lobe of the liver resting on the aorta.

Pulsation at the base of the heart may result from:

1. Aneurysm of the aortic arch.

2. Cardiac hypertrophy.

3. Shrinking of the lungs, as in tuberculosis.

Pulsation in the left axillary region may result from:

1. Large right-sided pleural effusions.

2. A tense, purulent effusion in the left pleural sac (pulsating empyema).

3. Aneurysm.

Unnatural pulsation in the carotids may result from:

1. Excitement of the heart from any cause.

2. Exophthalmic goiter.

3. Anemia.

4. Valvular disease, especially aortic regurgitation.

5. Aneurysm or dilatation of the vessels.

Jugular Pulsation.—The jugular vein often becomes distended in forced expiration and coughing. Inspiratory overfulness (Kussmaul's sign) of the jugular vein is sometimes noted in adherent pericardium.

A positive or systolic venous pulsation is observed in tricuspid regurgitation.

A pulsation may be transmitted to the jugular vein from the underlying carotid, but this false pulsation will still continue when light pressure is made on the vein at the root of the neck, while the true venous pulse will cease.

Precordial Prominence.—*Unnatural prominence of the precordium* may result from: (1) Enlargement of the heart;

(2) pericardial effusion; (3) aortic aneurysm; (4) rachitic deformity.

PALPATION

This serves to determine the position, force, extent, and rhythm of the apex-beat, and also to detect the presence of thrills.

A *thrill* is a vibratory sensation likened to that received when the hand is placed on the back of a purring cat. Thrills at the base of the heart may result from aortic stenosis, athroma of the aorta, aneurysm, and congenital heart disease (pulmonary stenosis and patency of the ductus arteriosus).

A systolic thrill may sometimes be felt at the apex in mitral regurgitation. A presystolic apical thrill is almost pathognomonic of mitral stenosis.

PERCUSSION

This serves to determine the shape and extent of the cardiac dulness.

The normal area of superficial or absolute percussion-dulness (the part uncovered by lung) is detected by light percussion, and extends from the fourth left costosternal junction to the apex-beat; from the apex-beat to the junction of the xiphoid cartilage with the sternum, and thence up the left border of the sternum.

The normal area of deep percussion-dulness (the heart projected on the chest-wall) is detected by firm percussion, and extends from the third left costosternal articulation to the apex-beat; from the apex-beat to the junction of the xiphoid cartilage with the sternum, and thence up the right border of the sternum to the third rib. The lower level of the cardiac dulness fuses with the liver dulness, and can rarely be determined by percussion.

The area of cardiac dulness is increased in: (1) Hypertrophy and dilatation of the heart. (2) Pericardial effusion. It

is apparently increased in shrinking of the lungs, as in tuberculosis.

The area of cardiac dulness is diminished in: (1) Emphysema. (2) Pneumothorax. (3) Pneumopericardium (rare). (4) Gaseous distention of the stomach.

AUSCULTATION

This serves to determine the quality, intensity, and rhythm of the heart-sounds, and to detect the presence of any adventitious sounds, such as murmurs. The two sounds heard over the heart have been represented by the syllables, "lubb, tup." The first sound (*systolic*) results from the muscular contraction of the heart and the closure of the auriculoventricular valves, and is synchronous with the apex-beat and carotid pulse. This sound is prolonged and dull. After the first sound there is a short pause, and then follows the second sound (*diastolic*), which results from the closure of the aortic and pulmonary valves. This sound is short and high-pitched. After the second sound a longer pause follows before the first is again heard.

The Intensity of the Heart-sounds.—*Both sounds are accentuated* in: (1) Excitement of the heart from any cause. (2) Anemia. (3) Cardiac hypertrophy. (4) Subjects with thin chest-walls. (5) Consolidation of the lung, as in tuberculosis and pneumonia.

Accentuation of the aortic second sound results from: (1) Hypertrophy of the left ventricle. (2) High arterial tension, as in chronic interstitial nephritis with arteriosclerosis. (3) Aortic aneurysm.

Weakening of the aortic second sound indicates weakness of the left ventricle.

Accentuation of the pulmonary second sound results from: (1) Pulmonary obstruction, as in emphysema, pneumonia, and the congestion of the lungs following mitral disease. (2) Hypertrophy of the right ventricle.

Weakness of the pulmonary sound indicates weakness of the right ventricle, and, occurring in diseases in which it should be accentuated, is of unfavorable omen.

Weakness of the mitral sound is noted in: (1) General obesity. (2) General exhaustion. (3) Degeneration or dilatation of the heart. (4) Pericardial or pleural effusion. (5) Emphysema.

Alteration in the Rhythm of the Heart-sounds.—*Reduplication of the Diastolic Sounds.*—This is probably due to a lack of synchronism in the closure of the aortic and pulmonary valves. It is frequently noted in health at the end of a long inspiration. Pathologic reduplication may occur whenever the pressure in either the pulmonary circulation or the peripheral arteries is abnormally increased. It is a common sign in mitral stenosis, emphysema, arteriosclerosis, and pericarditis.

Adventitious Sounds.—*Murmurs* are abnormal sounds produced in the heart or blood-vessels. They may result from: (1) Obstruction or regurgitation at the valves in consequence of valvular endocarditis. (2) Dilatation of the ventricle or relaxation of its walls, in consequence of which the auriculo-ventricular valves become relatively insufficient. (3) Roughening of the valves or of the intima of the great vessels. (4) Aneurysm (bruit). (5) Anemia (hemic murmur).

Exocardial murmurs are adventitious sounds of cardiac origin produced in the pericardium (pericardial friction-sound) or in the pleura or lung, adjacent to the heart (pleuropericardial friction-sound and cardiorespiratory murmur).

Pericardial Friction-sound.—This is an adventitious sound produced in pericarditis by roughening of the serous membrane. It is a harsh, grating, to-and-fro sound, quite superficial, often intensified by pressure with the stethoscope, and generally heard best in the fourth interspace near the sternum. It often varies in intensity from hour to hour, and is rarely transmitted beyond the precordial region.

Pleuropericardial Friction-sound.—This is a sound closely resembling the pericardial friction-sound, but produced by

inflammation of that part of the pleura that overlaps the heart. It is intensified by deep inspiration, and often disappears when the breath is held during expiration.

Cardiorespiratory Murmur.—This is a rare adventitious sound, produced by the rhythmic expulsion of air from the lappet of lung covering the heart by the cardiac contractions. The exact condition under which it occurs is not known. It is usually heard best at the end of inspiration, and is nearly always systolic in time. It is greatly modified by position, deep breathing, coughing, and holding the breath.

Aneurysmal Murmur, or Bruit.—In a large proportion of cases a murmur is heard in aneurysm. It is systolic in time, heard with greatest intensity over the sac of the aneurysm, and transmitted into the vessels of the neck. There is nothing in the character of the murmur to suggest its origin.

Hemic Murmurs.—Hemic murmurs have the following characteristics: They are soft and blowing in character; they are usually systolic in time; they are usually heard best over the pulmonary area; they are associated with evidences of anemia; they are not accompanied by signs of cardiac enlargement; they are often associated with a continuous hum in the veins of the neck; and they are more affected by deep breathing, position, and exercise than the murmurs of organic disease.

THE PULSE

The average frequency of the pulse in the healthy adult at rest is between 70 and 80. In new-born infants it is between 130 and 140, and in young children between 90 and 100.

Increased Frequency of the Pulse (Tachycardia).—Habitual frequency is sometimes noted in health. The frequency may be temporarily increased by erect posture, excitement, eating and the use of stimulants.

Abnormal frequency may be due to—(1) Pyrexia. The pulse usually bears a definite relation to the temperature, but in certain diseases, as scarlet fever and septicemia, it may

be disproportionately rapid, and in others, as meningitis and yellow fever, it may be disproportionately slow. (2) Hyperthyroidism. (3) Organic heart disease. (4) Severe anemias. (5) Reflex irritation, as in ovarian or uterine disease. (6) Essential paroxysmal tachycardia. (7) Action of certain drugs—belladonna, nitrites, thyroid extract, etc. (8) Rheumatoid arthritis (Sansom).

Infrequency of the Pulse (Bradycardia).—*Physiologic infrequency* is noted after fasting, sometimes in the puerperium, and habitually in certain persons (50 to 60 a minute).

Pathologic infrequency is observed in many conditions, notably—(1) In certain forms of organic heart disease (especially marked in lesions of the auriculoventricular bundle causing full heart-block). (2) In many toxemias—jaundice, uremia, myxedema, plumbism, digitalis-poisoning. (3) From pressure at the base of the brain sufficient to irritate the vagus, as in beginning meningitis, tumor, etc. (4) At the close of febrile diseases, as typhoid fever, pneumonia, etc. (5) In certain painful affections of the digestive tract. (6) In cachectic states.

Arrhythmia.—Alterations in the normal cardiac rhythm may depend upon disturbances of the nervous mechanism of the heart (*neurogenic arrhythmia*) or upon disturbances arising within the heart muscle itself (*myogenic arrhythmia*). If the irregular beats occur in a definite order the condition is termed *allorhythmia*. Extreme irregularity without any discernible order in the arrangement of the beats is frequently spoken of as *delirium cordis*.

Neurogenic Arrhythmia may be caused by strong emotions, by various toxic agents (tobacco, coffee, alcohol, bacterial poisons), by meningitis or cerebral lesions causing increased intracranial pressure, and by various impressions emanating from the organs, especially the stomach and bowel, and acting upon the heart in a reflex manner. In neurogenic arrhythmia, which is especially common in young nervous individuals, the irregularity is often closely related to phases of the respiration,

and usually all the beats are of the same strength and volume. It usually disappears when the cardiac rate is increased to 100 and is often abated by atropin.

Myogenic Arrhythmia.—Several types are recognized:

(1) *Extra-systoles.*—Extra-systoles are premature cardiac contractions arising from some abnormal site (ventricle, auricle, or, very rarely, auriculo-ventricular node), in the myocardium and due to increased myocardial excitability. The extra beat occurs from time to time following a normal beat at a shorter interval than the space between two of the normal beats. The interval following the extra-systole is longer than the average, and the succeeding regular beat is unduly strong and often manifest to the patient as a precordial thump. Irregularity in rhythm of the heart from this cause is very common. With quickening the pulse beyond 100 it usually becomes less marked or disappears.

Intermission of the pulse occurs when the refractory period (the period in which the ventricle is unresponsive to stimuli), following an extra-systole, overlaps the time of the next physiologic stimulation. True intermission must be distinguished from simulated intermission in which the ventricular contractions occur, but are too feeble at times to produce a peripheral pulse. A bigeminal pulse occurs when each normal systole is regularly followed by an extra-systole and a compensatory pause, and a trigeminal pulse occurs when a normal systole is regularly followed by two extra-systoles and a compensatory pause.

In rare instances the stimuli producing the extra-systoles arise in the auriculo-ventricular bundle (true nodal rhythm) causing the auricles and ventricles to contract almost simultaneously, as shown by the superposition of the auricular and ventricular waves (*a* and *c* waves) of the jugular pulse.

In many instances extra-systoles are not due to organic disease of the heart, but are caused by increased functional irritability of the myocardium, by the action of toxic agents (tobacco, tea, etc.), or by conditions acting reflexly on the heart,

(2) *Auricular Fibrillation*.—In this condition the uniform contractions of the auricle as a whole are replaced by a multitude of haphazard fibrillary contractions, and as a result the ventricular beats, and hence the pulse beats, become grossly irregular, both as to time and to force. The pulse rate is, as a rule, increased (110–150), but it may be nearly normal, or if heart-block coexists, even decreased. Other important signs are the absence of normal auricular waves in tracings taken from the jugular vein and from the cardiac apex, and in cases of mitral stenosis, the replacement of the rumbling presystolic murmur by a soft diastolic murmur. Symptoms of cardiac insufficiency are generally present, although some patients enjoy fair health for months or even years. Auricular fibrillation is the result of inflammatory or degenerative changes in the myocardium, and is especially common in cases of mitral stenosis. Once established, it is usually permanent, although in some instances it is paroxysmal. It occurs in more than 70 per cent. of all cases of heart failure with dropsy (Mackenzie).

(3) *Auricular Flutter*.—This condition is closely related to auricular fibrillation and arises from the same pathologic causes. It is characterized by an extremely rapid auricular rate (beyond 200 a minute), but the contractions are rhythmic and of uniform amplitude. It tends to persist indefinitely, although it is sometimes paroxysmal.

Auriculoventricular Heart-block.—This form of arrhythmia is due to the failure of the auricular contractions to reach the ventricle, owing to defective conductivity in the bundle of His. In complete heart-block the conducting function of the bundle is entirely lost and the ventricle develops an independent rhythm of its own, the pulse being usually regular and numbering about 30 per minute. In partial heart-block the conductivity of the bundle is merely impaired, the result being a prolongation of the interval between the auricular and the ventricular contractions (*a-c* interval) or the dropping of one ventricular beat in 6, 5, 4, etc. The diagnosis can always be made with instrumental aid and often without it; the asso-

ciation of bradycardia with a normal number of auricular contractions, as shown by inspection of the jugular vein, being characteristic. Heart-block may result from organic lesions in the conducting bundle, such as fibrosis, gumma, abscess, etc., from infectious toxemias or from the action of certain drugs, especially digitalis.

In some cases of complete heart-block the *Adams-Stokes syndrome* develops. This is characterized by a very infrequent pulse (5 to 40 per minute) and recurring attacks of a syncopal, epileptiform, or vertiginous character, probably the result of cerebral anemia. In the aged the Adams-Stokes syndrome is almost invariably an expression of degenerative myocarditis; in young adults it is usually indicative of syphilitic myocarditis, although in rare instances it may be of nervous origin.

Paroxysmal Tachycardia.—This condition is characterized by attacks of tachycardia with an abrupt onset and abrupt termination, and lasting from a few minutes to several days. Between the attacks the patient's health is usually good. The impulses exciting the contractions may originate in the sinoauricular node, the auricle, or, very rarely, the ventricle. The resemblance to paroxysmal auricular flutter may be close, but in the latter the rate is more rapid (in excess of 200), the onset and termination are much less abrupt, the attacks persist much longer, are influenced by digitalis, and are associated with definite evidence of myocardial disease. Paroxysmal tachycardia may sometimes be arrested by firm pressure over the right vagus, by deep breathing or holding the breath, vomiting, or the application of ice-bag to the precordium.

Pulsus Alternans.—In this condition there is a diminution in the contractile power of the heart resulting in the occurrence of alternating large and small beats. The pulse may be regular, but as a rule the small wave is delayed, so that the interval between the large beat and the small beat is greater than that between the small beat and the next large one. With extrasystoles the reverse occurs, that is, the small beat is premature. Pulsus alternans if persistent is of evil import.

Pulsus Paradoxus.—The characteristic feature of this pulse is a marked diminution in the size of the beats or an actual suppression of beats during full inspiration. It is probably due to some hindrance to the inflow or outflow of blood from the heart, caused by traction on the great vessels. It is not infrequent in adherent pericardium and disease of the mediastinum.

In many instances the type of cardiac irregularity present can be recognized by the palpating finger and the stethoscope, but very often the actual condition can be determined only with the aid of the polygraph or the electrocardiograph.

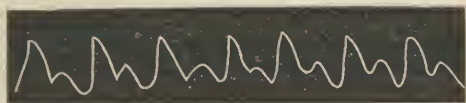


FIG. 6.—Sphygmogram of a dicrotic pulse.

The Dicrotic Pulse.—This is a pulse in which the main beat is quickly followed by a secondary wave or slight rebound of the vessel. It is frequently observed when the tension is low and the arteries are relaxed, as in exhausting fevers, such as typhoid.

Venous Pulse.—An abnormal (systolic) jugular pulse-wave is seen in tricuspid regurgitation. A venous pulse in the dorsum of the hand may be due to forcible propulsion of the blood through the capillaries, as in compensated aortic regurgitation, or to extreme relaxation of the arterioles, permitting the transmission of the pulse-wave, as in grave anemia.

Capillary Pulse.—This may be detected by the occurrence of systolic blushing in an area of the skin (preferably over the forehead) made hyperemic by friction, or in the everted lip which has been somewhat blanched by pressure of a glass slide. A capillary pulse is sometimes observed in aortic regurgitation, in severe anemia, and in exophthalmic goiter.

Asymmetric radial pulses may result from: (1) Anomalies in the distribution, size, or division of one of the vessels. (2) Aortic aneurysm. (3) An embolus or an atheromatous plate within the vessel. (4) Fractures, luxations, or inflammatory exudations causing compression of the vessel. (5) Compression of one vessel by tumors within or without the thorax.

"Water-hammer Pulse" (Corrigan's Pulse).—This pulse is characterized by a quick, powerful beat, which suddenly collapses or recedes. The peculiar pulsation may be distinctly visible, not only in the carotids, but throughout the brachial artery. This pulse is diagnostic of aortic regurgitation during the period of compensation, and its force is due to the excessive ventricular hypertrophy and to the large amount of blood expelled with each systole; its sudden recession is due to the incompetent valves failing to support the column of blood in the aorta.

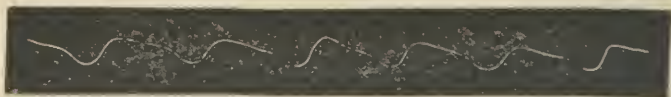


FIG. 7.—Pulsus tardus in aortic stenosis (Strümpell).

Tardy Pulse.—The wave rises gradually, is prolonged, and falls away slowly. In the tracing the ascending and descending limbs of the curve are sloping and the summit is blunt. A pulse of this type occurs in aortic stenosis.

The Blood-pressure.—This depends upon: (1) The strength of the heart; (2) the resistance in the vessels and their elasticity; and (3) the volume, and, to a less extent, the viscosity of the blood. Blood-pressure may be roughly estimated by noting the amount of force that is required to arrest the pulse at the wrist when the fingers are pressed upon the radial artery. It can be determined accurately only by means of the sphygmomanometer. The maximal blood-pressure in healthy persons at rest averages about 115 to 135 mm. for adults and about 90 to 110 mm. for children.

Abnormally high blood-pressure or hypertension (150 to 250 mm. or more) as a more or less transitory condition is observed in asphyxia, in certain intoxications (uremia, eclampsia, plumbism), and as a result of increased intracranial pressure; as a persistent condition, it may occur in chronic nephritis, in general arteriosclerosis, and as a functional disorder (essential hypertension, hyperpiesia). The last is most often seen in women at the time of the menopause.

Abnormally low blood-pressure or hypotension (110 to 80 mm. or lower) is observed in shock and collapse, acute infectious diseases, pulmonary tuberculosis, Addison's disease, many cases of acute and chronic cardiac disease, cachectic states, hemorrhage and conditions causing marked loss of body fluids, and after the administration of certain drugs, such as the nitrites, aconite, etc.

PALPITATION

Definition.—A rapid and tumultuous action of the heart perceptible to the patient. Rapidity not perceptible to the patient is not termed palpitation.

Etiology.—It may result from: (1) Reflex irritation, as from flatulent distention of the stomach. (2) Excitement, mental or physical. (3) Organic heart disease. (4) Exophthalmic goiter. (5) Overwork, as in the "irritable heart" of untrained recruits. (6) Anemia. (7) Neurasthenia or hysteria. (8) Paroxysmal tachycardia.

DROPSY

Definition.—An unnatural collection of serous fluid in the tissues or cavities of the body.

Etiology.—Dropsy may result from: (1) Chronic visceral affections that bring about venous stasis, as chronic heart disease, cirrhosis of the liver, and emphysema. (2) Local obstruction to the venous circulation by emboli, thrombi, tumors, etc. (3) Changes in the composition of the blood, as in anemia. (4) Increased permeability of the capillary walls, as in nephritis.

(5) Disturbed innervation, as in hysteria, angioneurotic edema and neuritis. (6) Excessive salt intake.

GENERAL CYANOSIS

Definition.—Blueness of the surface from insufficient oxidation of the blood.

Etiology.—Cyanosis results from: (1) Affections preventing the free entrance of air to the lungs, as in laryngeal or tracheal stenosis, asthma, emphysema, pneumonia; (2) obstruction to the venous return, as in uncompensated cardiac disease; (3) congenital heart disease, in which there is venous congestion or a direct admixture of arterial with venous blood; (4) erythremia (see p. 179); (5) conditions associated with methemoglobinemia, as acetanilid or antipyrin poisoning.

DISEASES OF THE HEART

PERICARDITIS

Etiology.—Inflammation of the pericardium is invariably a secondary process. (1) It may occur in infections generally, in particular rheumatism and chorea. (2) It may result from the extension of disease in adjacent structures, such as the lungs, pleuræ, mediastinal glands, ribs, etc. (3) It is not uncommon as a terminal condition in chronic diseases of various kinds, especially chronic nephritis. (4) It rarely follows local traumatism. The microorganisms most commonly concerned are the pyogenic cocci, the pneumococcus, and the tubercle bacillus.

Pathology.—The membrane becomes red, sticky and lusterless and in a short time a serofibrinous, fibrinous, or purulent exudation makes its appearance. In *serofibrinous pericarditis* there is, as a rule, only a slight fibrinous deposit, the exudation being composed chiefly of straw-colored fluid (from a few ounces to 2 pints or more). Occasionally the fluid is hemorrhagic, particularly in tuberculous cases. In the milder attacks the effusion may be completely absorbed, but in many cases adhesions form here and there between the surfaces and the pericardium becomes permanently thickened.

In *fibrinous pericarditis* little fluid is effused, but the heart is covered with a butter-like exudation, which ultimately undergoes organization and leads to partial or complete obliteration of the pericardial sac. Associated with obliterative pericarditis there are frequently adhesions between the external surface of the pericardium and surrounding parts, and also an increase of the fibrous tissue in the mediastinum (*indurative*

mediastinopericarditis. In other cases the pleuræ and the peritoneum share in the chronic inflammatory process, producing the condition described as *multiple serositis* (Pick's disease). Pericardial adhesions offer resistance to the ventricular contractions and ultimately cause hypertrophy and dilatation of the heart.

In *purulent pericarditis* from a few ounces to 2 or 3 pints of pus may be present in the sac. In rare instances recovery may occur, even without the evacuation of the pus, the latter becoming inspissated and finally infiltrated with lime salts.

In all forms of pericarditis the lesions extend, as a rule, a variable distance into the myocardium.

Symptoms.—Precordial pain or discomfort, palpitation, dyspnea, moderate fever, and weakness are the usual symptoms, but in many cases the disease is latent and only discovered on routine examination. The face may be unduly pale or distinctly cyanosed, the veins of the neck may be turgid, and occasionally, if the effusion is large, there may be hoarseness from pressure upon the recurrent laryngeal nerve or difficulty in swallowing from pressure on the esophagus.

PHYSICAL SIGNS.—In the early stage of serofibrinous pericarditis and throughout the fibrinous pericarditis the only characteristic sign is the friction sound. This is a superficial rubbing or creaking sound, usually double (to-and-fro) and of cardiac rhythm, but not absolutely synchronous with the normal cardiac sounds. It is best heard, as a rule, in the fourth left intercostal space and not transmitted beyond the precordium. Pericarditis with effusion is manifested by several definite signs:

Inspection.—The precordium may be abnormally prominent, especially in children.

Palpation.—The apex-beat is feeble or lost. A pulsation is sometimes apparent in the fourth interspace.

Percussion.—The area of cardiac dulness is increased and irregularly pear-shaped with the base directed downward and the stem upward. It often extends above the third costal

cartilage, a considerable distance to the left of the cardiac impulse, and into the right fifth intercostal space an inch or more to the right of the sternum (Rotch's sign), sometimes changing the cardio-hepatic angle, normally about 90 degrees, to an obtuse angle (Ebstein's sign). Other signs of variable constancy include change in the outline of dulness with change of the patient's position, unusual prominence of the sternal end of the first rib (Ewart), and an area of dulness with bronchial breathing near the angle of the left scapula (Bamberger's sign).

Auscultation.—The heart-sounds are indistinct (muffled) and often disproportionately feeble in comparison with the strength of the pulse. The friction sound sometimes persists.

Purulent Pericarditis.—The symptoms are similar to those of serofibrinous pericarditis, but septic phenomena—irregular fever, chills, sweats, pallor, and marked leucocytosis—are often present, and occasionally the precordial tissues are edematous. In doubtful cases recourse should be had to exploratory puncture.

Adherent Pericardium.—The diagnosis cannot always be made during life, but the following signs are suggestive. Enlargement of the heart with (1) systolic retraction in the region of the apex and also posteriorly in the region of the eleventh and twelfth ribs (Broadbent's sign); (2) inspiratory swelling of the cervical veins (Kussmaul's sign); (3) diastolic collapse of the cervical veins (Friedreich's sign); (4) absence of shifting of the apex-beat with change of the patient's posture and during full inspiration; (5) deficiency in the respiratory movements of the diaphragm; (6) the pulsus paradoxus (see p. 203). With these physical signs there are often symptoms of cardiac failure—dyspnea, edema of the legs, etc., or evidences of hepatic cirrhosis—ascites, splenic enlargement, etc. (see perihepatitis, p. 108).

Diagnosis.—*Acute Endocarditis.*—Endocardial murmurs have not the peculiar rubbing or creaking quality of friction sounds, are not affected by pressure with the stethoscope, are

heard most distinctly at valve points, are often propagated beyond the precordium, and have a definite relation to the sounds of the heart.

Cardiac Hypertrophy.—The enlargement develops slowly; the impulse is powerful; the apex-beat is displaced downward, and the cardiac sounds are loud.

Cardiac Dilatation.—In this condition there is frequently a wavy impulse over the precordium, the apex-beat is often displaced downward; the dulness is more globular in shape and rarely extends to the left beyond the cardiac impulse, upward above the third rib, or far into the right fifth intercostal space; the cardio-hepatic angle remains a right angle; the boundary between the dulness and pulmonary resonance is much less abrupt than in effusion; and, finally, the cardiac sounds, though weak, are usually clear.

Prognosis.—In the dry and serofibrinous forms the prognosis is good under favorable conditions. In the purulent form the outlook is extremely grave. The fibrinous form, though not immediately fatal, is very serious on account of the secondary changes that it induces in the cardiac muscle.

Treatment.—Absolute rest is imperative. The diet should be light, but nutritious. Locally, an ice-bag is serviceable. Leeching is beneficial in robust subjects. Blisters are useful when there is great pain. In rheumatism salicylate treatment should be continued. Opium is often necessary to secure rest and to allay pain. If heart-failure occurs, such stimulants as whisky, strychnin, digitalis, and caffein must be employed.

Pericardial Effusion.—If the effusion is serous, absorption may be aided by the application of small blisters, by the administration of diuretics—digitalis, caffein, theobromin—and by the administration of saline purges. Potassium iodid is of doubtful efficacy. Diaphoretics, particularly pilocarpin, should not be used. If pressure symptoms become urgent or the effusion does not yield after a thorough trial to the measures just mentioned, paracentesis should be performed. The puncture may be made in the left fifth intercostal space about

an inch from the sternum, or in the left costosternal angle, the needle being directed upward and backward.

In pericarditis with purulent effusion the indications are to incise the sac and to afford the freest possible outlet for the pus. The mortality is high (about 60 per cent.) because of the associated conditions. In adherent pericardium the treatment is that of chronic valvular disease. In cases with marked systolic retraction of the precordial region resection of the underlying ribs and costal cartilages (cardiolysis) has been done with some success.

OTHER AFFECTIONS OF THE PERICARDIUM

Hydropericardium (dropsy of the pericardium) results from the causes of general dropsy, especially heart disease or nephritis. The physical signs are those of serofibrinous pericarditis in the stage of effusion.

Hemopericardium (blood in the pericardium) may result from traumatism, the rupture of an aneurysm, or the rupture of the heart itself. In cancerous and tuberculous pericarditis the serous exudate is often more or less bloody.

Pneumopericardium (air in the pericardium) is very rare. It may result from trauma or the rupture of an adjacent air-containing organ. Thus it may be produced by pneumopyothorax, a phthisic cavity, or ulceration of the esophagus or stomach. The symptoms are those of pericarditis. The characteristic physical signs are the occurrence of tympanitic and dull areas in the precordial region changing in relation with changes in the patient's posture and a loud churning murmur ("mill-wheel" sound) on auscultation.

ENDOCARDITIS

(Valvulitis)

Definition.—Inflammation of the lining membrane of the heart. The process is usually confined to the valves.

Varieties.—(1) Simple, benign, or verrucose endocarditis. This begins as an acute affection, but usually leads to chronic sclerotic valvular disease. (2) Malignant or ulcerative endocarditis. No sharp line, either clinically or anatomically, can be drawn between this and simple endocarditis. The terms malignant and ulcerative are used to designate a more intense

infection. (3) Chronic or sclerotic endocarditis. This may be the continuation of acute endocarditis or it may be chronic from the outset.

Etiology.—*Acute endocarditis* usually results from acute articular rheumatism, chorea, one of the infectious fevers, or septicopyemia. Gonorrhea, arthritic purpura and acute tonsillitis are less common causes. At least 60 per cent. of all cases of acute articular rheumatism are complicated with endocarditis. The young are more liable to be attacked than the aged. Sixty-two of 73 fatal cases of chorea collected by Osler showed endocarditis. In rheumatism and chorea the mitral valve is most frequently affected and the valves of the right heart are seldom involved. Of the infectious fevers, scarlatina and pneumonia are most prone to heart complications.

The *malignant* type, which may be acute or subacute, is prone to develop on valves already the seat of chronic inflammation, but it may be primary. It usually follows septicopyemia, pneumococcic infection, a specific fever, gonorrhea, or focal infection, especially in the tonsils. Occasionally, it occurs in rheumatism or chorea.

The microorganisms most frequently detected in the lesions of acute endocarditis are the streptococci, staphylococci, pneumococcus and the gonococcus.

Chronic endocarditis may be congenital, may follow an acute attack, or may develop insidiously as a sequence of syphilis, gout, alcoholism, plumbism, or chronic nephritis. Overuse of the muscles favors its occurrence, and like arteriosclerosis, which it often accompanies, it may be an expression of senility.

Pathology.—Postnatal endocarditis most commonly involves the valves of the left side of the heart. Prenatal endocarditis most commonly involves the valves of the right side of the heart.

In simple endocarditis the surface of the valve becomes red, swollen, and lusterless. Later, a row of bead-like vegetations (thrombi) appears along the line of maximum contact, which is about 2 mm. from the free margin of the valve. Microscopically, the endothelium beneath the vegetations shows

evidence of necrosis, and the adjacent tissue, a round-cell infiltration. The vegetations may be whipped off by the blood-current and carried to distant organs, as the brain, kidney, or spleen; but in the vast majority of cases, if life is preserved, they are transformed, together with the cellular exudate, into fibrous tissue (*chronic endocarditis*), which not only thickens the valves, but, by contracting, so shortens and distorts them that they are rendered in one instance obstructive to the onward flow of blood, and in another incompetent to close the orifice over which they preside. Finally, retrograde changes ensue, the thickened valves becoming fatty and calcareous.

The myocardium is probably more or less involved in every case of endocarditis.

The *malignant* type is characterized by luxuriant vegetation and more or less extensive necrosis and ulceration of the underlying tissues. Destruction of valve leaflets, chordæ tendinæ, etc., are not uncommon sequels and in some cases the inflammation spreads to the cardiac walls (mural endocarditis) or to the great vessels. Embolic showers frequently result from disintegration of the vegetations.

Symptoms.—In many cases of *simple acute endocarditis* there are no subjective disturbances and the only evidence of a valvular lesion is an alteration in one of the heart sounds (most commonly the first sound at the apex), which gradually develops into a definite murmur. In some cases, however, an increase in the fever, cardiac overaction out of proportion to the degree of temperature, irregularity of the pulse, precordial distress and dyspnea are also present.

In *malignant endocarditis* the general symptoms may be those of a frank septicopyemia—irregular fever, sweats, chills, progressive anemia and emaciation, leukocytosis, etc.—or the picture may be more like that of typhoid fever or of acute meningitis. Symptoms and physical signs referable to the heart, such as an increased pulse-rate, palpitation, precordial distress, dyspnea, and a murmur at one or other of the valve-points, are usually, but not invariably, present. Marked

variations in the intensity and character of the murmurs from day to day are often noted and are significant. Embolic phenomena occur sooner or later in a larger proportion of cases and are of the greatest diagnostic value. Lodging in the skin emboli may cause a petechial eruption (especially significant); in the kidney, lumbar pain and hematuria; in the spleen, painful enlargement of the organ; in the lung, hemoptysis, with localized dulness and fine rales; in the brain, aphasia, hemiplegia or monoplegia; in the eye, retinal hemorrhage or optic neuritis; and in the large vessels of the limbs, gangrene or infective aneurysms. In doubtful cases blood cultures may afford valuable diagnostic aid.

Malignant endocarditis usually lasts from two to six or eight weeks, but in some instances the infection is more subdued and continues for many months (*chronic or subacute infective endocarditis*). This form is most commonly associated with the *streptococcus viridans*, and is characterized by remittent or intermittent fever, progressive anemia and weakness, petechiæ in the skin or mucous membranes, embolic phenomena, and, not rarely, by the occurrence of fugitive tender nodules in the subcutaneous tissue.

In *chronic sclerotic endocarditis* the symptoms are entirely due to the mechanical effects caused by leaks or obstruction at the valves and to weakening of the heart muscle (see chronic valvular disease, page 215).

Diagnosis.—*Typhoid Fever.*—The gradual onset, the more regular fever, the abdominal symptoms, the roseolar rash, the Widal reaction, the bronchial catarrh, the early enlargement of the spleen, and the absence of leukocytosis and of embolic phenomena will serve to separate typhoid fever from malignant endocarditis.

Malarial Fever.—This may be recognized by the presence of the malarial parasite in the blood and by the absence of leukocytosis and cardiac signs.

Prognosis.—Acute simple endocarditis does not often prove fatal, but it rarely leaves the valve undamaged. Under

favorable conditions, however, compensatory hypertrophy of the heart ensues and good health may be preserved for an indefinite period. Rapid dilatation of the heart indicates concurrent myocarditis and is a serious sign. Malignant endocarditis almost always proves fatal, but occasionally recovery ensues with more or less permanent damage to the heart.

Treatment.—The treatment of acute endocarditis is mainly that of the causal condition. *Prolonged and complete rest* is of the greatest importance. The patient should be confined to bed not only during the attack, but for several weeks after it has subsided, in order to allow sufficient time for the damage to be repaired or for compensatory hypertrophy to be established.

Externally, an ice-bag is often useful in allaying excitement of the heart. Mild mercurial or saline aperients may be used from time to time for their depurative effect. Digitalis may be of service if the pulse is weak and irregular, but generally it is not indicated. Heart-failure is to be combated by such stimulants as alcohol, ammonia, strychnin, and caffeine. Repeated vesication and the prolonged use of potassium iodid have been warmly advocated.

CHRONIC VALVULAR DISEASE

PERIOD OF COMPENSATION

Compensation is effected by an increase in the strength and size of certain cardiac chambers sufficient to enable the arterial system to receive its normal supply of blood, notwithstanding obstruction or regurgitation at one or more of the valves. The duration of this period is indefinite, and depends largely upon the nature and extent of the valvular defect, the amount of damage sustained by the cardiac muscle, and the hygienic conditions to which the patient is subjected. During the period of compensation the existence of a valvular defect may be revealed only by physical signs, and a lessened response

of the heart to demands for increased activity, subjective disturbances being trivial or imperceptible.

Aortic Stenosis.—*Definition.*—Obstruction to the flow of blood into the aorta from thickening or adhesion of the aortic segments. Uncomplicated aortic stenosis is a rare lesion. It occurs usually in elderly persons as a part of general arteriosclerosis.

PHYSICAL SIGNS.—*Inspection.*—In many cases, the apex-beat is forcible and is displaced downward and to the left.

Palpation confirms inspection, and often detects a systolic thrill at the base of the heart.

Percussion usually reveals an increased area of cardiac dullness, especially in the long axis of the heart.

Auscultation.—A harsh systolic murmur is heard at the base of the heart and propagated into the carotids.

Pulse.—The pulse is characteristic. It is small, rises and falls very slowly (*pulsus parvus et tardus*), and is fairly tense.

Compensation and Sequences.—Interference with the discharge of blood into the aorta is followed by hypertrophy of the left ventricle. Ultimately, when the myocardium becomes enfeebled, dilatation ensues and leads to imperfect closure of the mitral orifice, that is, to relative mitral insufficiency.

Diagnosis.—The harshness of the murmur, weakness of the second sound, palpable thrill, enlargement of the left ventricle, and especially the characteristic pulse will serve to distinguish aortic stenosis from other conditions causing systolic murmurs in the aortic area, such as anemia and atheroma of the aorta.

Aortic Insufficiency.—*Definition.*—Failure of the aortic valves to prevent the return of blood into the left ventricle because of (1) shrinking of the leaflets, the result of endocarditis or sclerosis; (2) rupture of the leaflets from mechanical strain or ulceration (comparatively rare); or (3) enlargement of the aortic ring from dilatation or aneurysm of the aortic arch. The sclerotic form is the most common. Syphilis, rheumatism, and physical overexertion are the chief etiologic factors. Oc-

curing as an isolated valvular lesion between the ages of twenty five and fifty, it is usually the result of syphilis.

PHYSICAL SIGNS.—*Inspection.*—The cardiac impulse is diffuse and forcible and the apex-beat is displaced downward and outward. The peripheral arteries, especially the carotids, often throb violently, and in many subjects there is bulging of the precordium.

A capillary pulse is present in the majority of cases. It is shown in the alternate blushing and paling, which occurs when slight pressure is made on a finger nail or a glass slide is pressed upon the lip. Occasionally, pulsation may be observed in the superficial veins on the back of the hand.

Percussion reveals a marked increase in the area of cardiac dullness, especially toward the left and downward.

Auscultation.—There is a diastolic murmur, most distinct at the base of the heart, in the second or third interspace, near the right or left border of the sternum, and transmitted down the sternum. The aortic second sound is usually suppressed. In the majority of cases a systolic murmur is also heard at the aortic area. In some instances a presystolic murmur (Flint murmur) is audible at the apex. It is probably due to the impact of the regurgitating blood stream upon the anterior mitral leaflet. Auscultation over the large arteries, especially the femorals, sometimes reveals a sharp systolic shock ("pistol-shot" sound), and if slight pressure be made with the stethoscope, a double murmur (Duroziez's sign).

The *pulse* is characteristic and is known as the Corrigan or water-hammer pulse. The wave is very large, with a quick upstroke and sudden fall. It is best perceived by elevating the arm and grasping the forearm above the wrist. The systolic pressure is often above normal and the diastolic pressure very low; moreover, the systolic pressure is usually much higher in the legs than in the arms.

Compensation and Sequences.—The increased pressure within the left ventricle during diastole results in marked dilatation and hypertrophy of that chamber, the heart in

consequence becoming enormously enlarged (*cor bovinum* or ox-heart). Ultimately, relative mitral insufficiency ensues from stretching of the mitral ring.

Mitral Stenosis.—*Definition.*—Obstructive to the flow of blood through the mitral orifice, the result of thickening or fusion of the valve-leaflets. Mitral insufficiency often coexists. The disease may follow acute endocarditis or develop gradually in association with arteriosclerosis. The endocarditic form, the more common form, is observed chiefly in the young.

PHYSICAL SIGNS.—*Inspection.*—The apex-beat is, as a rule, not much displaced. Some bulging is often noticed over the lower part of the sternum.

Palpation.—In the majority of cases a very characteristic purring thrill is to be felt in the region of the apex. It is diastolic or presystolic in time, sharply localized, and ends abruptly with the apex-beat.

Percussion usually reveals an increase in the cardiac dulness chiefly to the right of the sternum.

Auscultation.—In compensated cases a rumbling or vibratory murmur is heard a little to the right of the apex late in diastole (presystolic) or throughout diastole. This murmur is confined, as a rule, to a small area, becomes louder toward the close of the diastole (crescendo-like), and ends abruptly in a loud, snapping first sound. It disappears when auricular fibrillation occurs. The second pulmonic sound is usually accentuated. The *pulse* is in no way characteristic. It becomes very irregular, however, with the occurrence of auricular fibrillation (see p. 201).

Compensation and Sequences.—The first strain is upon the left auricle, which becomes enormously enlarged. With over-distention of the left auricle the intrapulmonary pressure is increased and this results in hypertrophy or dilatation of the right ventricle. Ultimately, relative tricuspid insufficiency is likely to ensue from stretching of the tricuspid ring. Unless mitral insufficiency is also present the left ventricle is not usually enlarged.

Mitral Insufficiency.—*Definition.*—Defective closure of the mitral valve owing to enlargement of the mitral ring, the result of weakening or dilatation of the left ventricle (relative insufficiency), or to contraction and curling of the mitral leaflets, the result of endocarditis or slowly progressing sclerosis. Mitral insufficiency is the most common of the valvular defects.

PHYSICAL SIGNS.—*Inspection.*—The apex-beat is displaced outward rather than downward. Bulging of the precordium and a diffuse pulsation are often noted.

Percussion.—The area of cardiac dulness is increased transversely, both to the left and to the right.

Auscultation reveals a systolic murmur, loudest near the apex, and transmitted to the axilla and sometimes to the angle of the scapula. The pulmonic second sound is often accentuated.

The *pulse* shows no characteristic changes.

Compensation and Sequences.—Enlargement of the left auricle, increase in the intrapulmonary pressure, and hypertrophy and dilatation of the right ventricle are brought about in the same way as in mitral stenosis. Hypertrophy and dilatation of the left ventricle also occur in response to the large amount of blood discharged into this chamber from the auricle during each diastole. With weakening of the right ventricle relative tricuspid insufficiency is likely to ensue.

Tricuspid Stenosis.—This lesion is rare and frequently mistaken for mitral stenosis, with which it is usually associated. The area of cardiac dulness is increased to the right, and there is a presystolic murmur of maximum intensity over the lower end of the sternum. Marked cyanosis is usually present.

Tricuspid Insufficiency.—The organic form, the result of inflammation or degeneration of the tricuspid leaflets is comparatively rare, but the relative form due to enfeeblement or dilatation of the right ventricle, the result of obstruction in the pulmonary circulation (mitral lesions, emphysema, etc.), primary myocardial disease, or general malnutrition is com-

mon. Important signs are cyanosis; distention of the superficial veins; a pulsation in the jugular veins, synchronous with the heart-beat; systolic expansile pulsation of the liver; an increase in the area of cardiac dulness particularly to the right of the sternum; and a blowing systolic murmur of maximum intensity over or near the lower third of the sternum.

Pulmonary Stenosis and Pulmonary Insufficiency.—These lesions are very rare. *Pulmonary stenosis* is usually congenital. Important signs are enlargement of the heart to the right; a systolic thrill in the second left interspace near the sternum; a systolic murmur in the same area, which in contrast to the murmur of aortic stenosis, is not transmitted into the carotid arteries; and suppression or extreme weakness of the pulmonic second sound. In the congenital cases cyanosis is usually present from birth.

The physical signs of *pulmonary insufficiency* resemble those of aortic insufficiency. In the former, however, the heart is enlarged to the right, the aortic second sound is usually distinct and the Corrigan pulse is absent.

PERIOD OF DECOMPENSATION

Decompensation may result from: (1) Increasing damage to the valves or heart muscle, as in lesions of a degenerative type; (2) senility, leading to arterial and cardiac degeneration; (3) intercurrent disease (influenza, pneumonia, acute nephritis, etc.), throwing additional strain upon the heart; (4) undue physical exertion; (5) pregnancy and parturition; (6) mental shock.

Symptoms.—The symptoms are varied and in general are due to venous stasis, local anemia, accumulation of carbon dioxid, toxemias from associated renal or hepatic disease, or gross secondary lesions, such as hydrothorax, ascites, or infarct of the lungs. Dyspnea is the most frequent symptom. It may be constant or only present on exertion. In many cases it is worse during the night. Asthmatic attacks and Cheyne-Stokes breathing may also occur. Cyanosis is some-

times a conspicuous feature. Cough and expectoration are not uncommon, and as a result of intense pulmonary engorgement or of pulmonary infarct hemoptysis may occur. Edema appears at some period in most cases. It usually begins in the feet and spreads upward, often involving the serous sacs. The right pleura is more often affected than the left.

Gastrointestinal disturbances are frequently present and with failure of the right heart the liver becomes enlarged and tender, and slight jaundice not rarely develops. The urine is usually scanty and albuminous, and may contain erythrocytes and tube casts. Many patients complain of palpitation and of precordial discomfort or pain. True angina pectoris may occur. Cerebral disturbances, such as headache, vertigo, faintness, insomnia, delusions and hallucinations, are not infrequent. The pulse is weak and irregularities due to extra-systoles, auricular fibrillation, or heart-block (see p. 201) are very often noted.

In *mitral cases* the pulmonary features (cough, expectoration, hemoptysis, etc.), cyanosis and edema are usually the dominant symptoms. In *aortic cases* precordial pain, pallor (aortic facies), and cerebral disturbances are especially common. Edema is rarely pronounced and embolic phenomena are uncommon. In *tricuspid insufficiency* orthopnea, gastric disturbances, painful swelling of the liver and ascites are usually conspicuous features.

Prognosis of Chronic Valvular Affections.—The character and intensity of the murmur are of little moment in determining the gravity of the lesion. Much depends upon the nature, site and grade of the valvular defect, the state of the myocardium, the general health of the patient and his age, habits and social condition. The relative gravity of the various lesions, in order from the most to the least serious, is probably as follows: Tricuspid insufficiency, aortic insufficiency, mitral stenosis, aortic stenosis, mitral insufficiency. With a stationary lesion and ample compensation, mitral insufficiency is not incompatible with a long life free from discomfort. Sudden death occurs

most frequently in aortic regurgitation. Unfavorable factors, generally speaking, are early childhood, marked enlargement of the heart, dyspnea upon slight exertion, irregular rhythm, the likelihood of a recurrence of the causal factor, lesions in the arteries or other viscera, poor general health and bad habits.

Treatment.—If compensation is well maintained, the treatment should be purely hygienic.

Stage of Decompensation.—Rest, both physical and mental, is the most important element in the treatment. The diet should be nutritious, but readily digestible. In some cases it may be well to restrict the diet for a time to milk, giving 3 or 4 ounces every two hours. When dropsy is present it may be desirable to limit the intake of liquids and to withhold salt from the food. The most reliable cardiac stimulant is digitalis (10 to 30 minims of the tincture two or three times a day). It may be given in any form of valvular disease when there are dyspnea, edema, deficient urination, and a frequent, weak, irregular pulse. It is of the greatest service in auricular fibrillation. Strophanthus rarely succeeds when digitalis fails. Strychnin is the most valuable adjuvant to digitalis, especially if there are degenerative changes in the heart. Mercurial and saline aperients are useful in lowering venous tension, and without their aid digitalis may fail. If the right ventricle is greatly overdistended and cyanosis is marked, venesection to the extent of half a pint or more may prove life-saving. Iron and arsenic are very serviceable when there is anemia. They may sometimes be combined advantageously with digitalis and strychnin, as in the following pill:

R. Arseni trioxidi..... gr. $\frac{1}{2}$
 Massæ ferri carbonatis..... gr. xx
 Strychninæ sulphatis..... gr. ss
 Pulveris digitalis..... gr. xx—xxx.—M.

Fiant pilulæ No. xx.

SIG.—One pill after meals.

Dropsy.—The most useful measures are hydragogue cathartics (salines in concentrated solution; compound jalap

powder, 30 to 40 grains; and elaterium, $\frac{1}{8}$ grain); diuretics (digitalis, caffein, theobromin, theocin, vegetable salts of potassium); the application of smooth, firm bandages to the limbs; and the introduction of fine silver cannulæ (Southey's tubes), or incisions behind the ankles. Hydrothorax and ascites call for tapping, especially if the breathing is much embarrassed.

Restlessness and Insomnia.—On the whole, morphin ($\frac{1}{4}$ – $\frac{1}{6}$ grain) with atropin ($\frac{1}{150}$ – $\frac{1}{120}$ grain), preferably hypodermically, is the best sedative. Chloralamid, bromids, barbital and trional are worthy of confidence.

Pain.—Temporary oppression is often relieved by warm or cold applications and the administration of Hoffmann's anodyne. Severe continuous pain may yield to leeching or blistering. In anginoid pains nitrites and potassium iodid are often efficacious. For nocturnal distress morphin is especially useful.

Sudden heart-failure must be met by the administration of diffusible stimulants, such as ammonia and alcohol. The intravenous injection of strophanthin ($\frac{1}{120}$ – $\frac{1}{60}$ grain) often acts very satisfactorily. It should not be employed, however, if digitalis has recently been used, or repeated within forty-eight hours, since serious cumulative effects may ensue. The application of heat to the precordium is useful.

After-treatment.—The important measures are moderation in diet, regular care of the bowels, adequate rest alternating with mild exercise, warm, but not, hot saline baths, and mild frictional massage.

ENLARGEMENT OF THE HEART

Varieties.—(1) *Simple Hypertrophy.*—The muscle of the heart is increased in thickness, but the cavities are of normal size.

(2) *Eccentric Hypertrophy (Hypertrophy with Dilatation).*—The muscle is thickened and the cavities are increased in size.

(3) *Simple Dilatation.*—The muscle is thinned and the cavities are increased in size.

Etiology.—*Hypertrophy* is usually the result of increased demands upon the functions of the heart. Thus, it may be due to—(1) Valvular disease; (2) abnormal resistance in the peripheral circulation, as in arteriosclerosis and chronic renal disease (left ventricle); (3) abnormal resistance in the pulmonary circulation, as in emphysema and cirrhosis of the lung (right ventricle); (4) prolonged exertion, as in athletes; (5) long-continued palpitation or tachycardia, as in exophthalmic goiter or tobacco heart; (6) interference with the ventricular contractions by pericardial adhesions.

Dilatation of the heart results from the same causes. It is more likely to occur than hypertrophy when the demands are sudden and severe, or when they fall upon a heart the walls of which are already degenerated.

Pathology.—In *hypertrophy* the muscle of the heart is firm and of a dark-red color. The normal weight (8 or 9 ounces) may be doubled or trebled. When the left ventricle is chiefly involved, the organ is increased in length. When the right ventricle is chiefly involved, the organ becomes more globular. Microscopically, the fibers are increased in size and in number.

In *dilatation* the heart muscle is softer, more flabby, and often lighter in color from degenerative changes.

Symptoms.—*Hypertrophy.*—Unless the hypertrophy is more than compensatory, no symptoms result. Excessive hypertrophy may give rise to precordial distress and symptoms of cerebral hyperemia—headache, tinnitus aurium, flashes of light, etc.—and the following physical signs: bulging of the precordium; a heavy impulse; displacement of the apex-beat downward and to the left; an increase in the area of cardiac dulness; a loud, booming first sound; accentuation of the aortic second sound or of the pulmonic second sound, according as the hypertrophy involves the left or right ventricle; and a strong, full pulse.

Dilatation also gives signs of cardiac enlargement, but the impulse is feeble or imperceptible, the first sound is short and weak (clicking), the pulse is rapid and feeble, and often

irregular or intermittent, and usually there are symptoms of venous congestion—dyspnea, cough, edema, flatulent dyspepsia, and deficient urination. Soft systolic murmurs, the result of relative mitral or tricuspid insufficiency, may be heard.

Treatment.—In *hypertrophy* treatment is rarely called for.

The treatment of *dilatation* is that of decompensation in valvular disease.

ACUTE MYOCARDITIS

Definition.—Acute inflammation of the heart muscle.

Etiology.—It results from the same cause as acute endocarditis.

Pathology.—It is usually associated with endocarditis or pericarditis, but sometimes the myocardium is the only part of the heart affected. The inflammatory process is always accompanied with more or less parenchymatous or fatty changes in the muscle-fibers. The characteristic feature is the infiltration of the interstitial tissue with round cells.

Symptoms.—The symptoms are often masked by the primary disease. Dyspnea, precordial discomfort, palpitation, pallor, and weakness of the pulse out of proportion to the severity of the general infection are important manifestations. The pulse is usually rapid and irregular, but there may be bradycardia from vagal disturbance or depressed conductivity of the auriculo-ventricular bundle (heart-block). The first sound at the apex is weak and indistinct or replaced by a soft systolic murmur, and the blood-pressure is, as a rule, low. The heart may or may not be dilated.

Treatment.—The treatment is that of acute endocarditis. The most useful stimulants are ammonia, whiskey, camphor, adrenalin, strychnin and caffein. Digitalis may be of service, but its effects must be carefully noted.

CHRONIC MYOCARDITIS

This term is used to designate various retrograde changes (fatty degeneration, fibrosis, atrophy of fibers) in the heart muscle, resulting in impairment of cardiac efficiency.

Etiology.—The disease often develops coincidentally with endocarditis or pericarditis and from the same causes; it may be secondary to sclerosis of the coronary arteries; it may occur as an independent condition, the causal factor being focal infection, syphilis, alcoholism, gout, hyperthyroidism, chronic nephritis or a wasting disease, such as diabetes or leukemia.

Symptoms.—Dyspnea, appearing at first only on exertion, is an almost constant symptom. Palpitation or discomfort in the region of the heart frequently accompanies the dyspnea. Digestion may be good, but not rarely patients complain of a sense of weight in the epigastrium and flatulence. Progressive pallor and weakness often develop from increased venous tension and interference with absorption. Paroxysms of angina pectoris, of the Stokes-Adams syndrome (see p. 202), or of acute pulmonary edema (cardiac asthma) may occur. In many cases the symptoms of chronic nephritis form a part of the clinical picture (*cardiorenal group*), the renal condition standing in the relation of either cause or effect to the cardiac inadequacy. In the absence of valvular lesions, the evidences of general venous engorgement, such as edema of the legs, hydrothorax and enlargement of the liver, are usually late in appearing. Toward the end the breathing may become of the Cheyne-Stokes type.

The heart usually presents some degree of enlargement. The first sound at the apex is often indistinct or replaced by a soft systolic murmur. The aortic second sound may be relatively accentuated. The pulse is variable. It may be abnormally frequent or infrequent. In many cases it is weak, but not rarely for a considerable period it is of high tension, the manometer registering from 160 to 200 mm. or more. With increasing myocardial incompetence, some form of arrhythmia (see p. 199) usually develops.

Prognosis.—The prognosis is always more or less serious, and carries with it the possibility of sudden death; nevertheless, with care the patient may live many years.

Treatment.—Muscular overexertion, mental strain, and excitement, as far as possible, should be avoided. The diet must be simple and readily digestible. Alcohol should be avoided and tobacco used sparingly, if at all. If the pathologic changes are not far advanced, graduated exercise, with warm saline baths, as in the well-known Nauheim treatment, may be efficacious. Constipation should be relieved. A change of climate is sometimes helpful, but high altitudes must be avoided.

Among special remedies, digitalis holds the first place. Strychnin ($\frac{1}{60}$ – $\frac{1}{40}$ grain, thrice daily) is often a useful adjuvant. If there is anemia arsenic and iron may be of value. In premature arteriosclerosis, especially if there are signs of syphilis, iodids may prove beneficial. For nocturnal attacks of cardiac asthma morphin is indispensable. Nitrites are useful in relieving and preventing anginoid attacks and in lowering the arterial tension when it is excessively high. Dropsy, insomnia, and sudden heart failure will require the same treatment as when occurring in decompensated valvular disease (see p. 222).

FATTY HEART

This term is applied to both fatty infiltration and fatty degeneration of the heart.

Fatty Infiltration.—This consists in an excess of the fat which is normally present in variable amounts beneath the epicardium, especially along the blood-vessels and in the grooves. In advanced cases the fat is also found between the muscle-fibers. The latter may remain normal for a long period, but ultimately, owing to compression, they undergo atrophy and fatty degeneration. Fatty infiltration results from the causes which lead to general obesity.

The *symptoms* are rarely marked unless the muscle-fibers themselves are affected. It may be suspected in obese subjects when there is complaint of dyspnea and palpitation upon

slight exertion, and the heart sounds are indistinct and the pulse is small and weak.

In the early stages the *treatment* is that of obesity—regulation of diet, restriction of liquids, graduated exercise, Nauheim baths, etc. In advanced cases the treatment is that of fatty degeneration of the heart.

Fatty Degeneration.—This is a degeneration of the muscle-fibers themselves, with the deposition of fat. It is frequently due to local anemia from sclerosis of the coronary arteries; it follows hypertrophy in valvular disease; it is a common result of malnutrition from old age, wasting diseases, or grave anemia; it is associated with parenchymatous degeneration (cloudy swelling) in severe infections; it occurs also in acute mineral poisoning, as by phosphorus, antimony, or arsenic.

The muscle of the heart is pale, soft, and flabby, and feels greasy to the touch. Microscopically, the fibers are found filled with small, dark fat-granules.

The *symptoms* are much the same as those of chronic myocarditis and the condition requires similar *treatment*.

ANGINA PECTORIS

(Neuralgia of the Heart; Stenocardia)

Definition.—A symptomatic affection most commonly associated with occlusion of the coronary arteries and degeneration of the myocardium, and characterized by severe paroxysmal pain in the region of the heart.

Etiology.—It usually develops after middle life, and is very much more common in men than in women. The predisposing causes are those of arteriosclerosis. In some instances an hereditary tendency has been noted, and not infrequently the attacks have been preceded by prolonged mental anxiety.

A false form of angina (*pseudo-angina*), rarely, if ever fatal, sometimes occurs in association with hysteria, vasomotor disturbances or the excessive use of tobacco.

Pathology.—Disease of the coronary arteries, sclerotic aortitis and myocardial degeneration are the lesions usually found after death. Occasionally, typical attacks occur in lesions of the aortic valve, especially insufficiency, and in aortic aneurysm.

Symptoms.—The attacks are usually excited by strong emotion, muscular effort, exposure to cold, or flatulent indigestion, and are characterized by intense pain, radiating from the heart to the shoulder and arm (usually the left), a sense of impending death, dyspnea, and a pale, anxious face. The pulse is very variable. The attacks last from a few seconds to several hours. Death may occur in the first attack, or there may be recurring attacks over a period of many years.

Hysteric Angina.—This neurosis is seen chiefly in women, whereas true angina is rare in women; there is no evidence of organic heart disease; the attack is often nocturnal, and is longer in duration; emotional outbreaks, such as moaning and crying, are common, and other stigmata of hysteria are usually present.

Gastralgia.—The pain is apt to appear when the stomach is empty, and is relieved by stimulating food; it does not radiate to the shoulder and arm; there is no sense of impending death, and no evidence of structural heart disease.

Prognosis.—True angina is always of grave import. Sudden death may occur at any time. In false angina the prognosis is favorable.

Treatment.—The general treatment is that of chronic myocardial disease. In syphilitic cases specific medication is, of course, indicated. Abstinence from alcohol and tobacco should be strictly observed. The most valuable special remedies, in the order of their efficacy, are the nitrites, iodids, theobromin, and arsenic.

The Attack.—No drug is so generally useful as amyl nitrite (3 to 5 minims on a handkerchief). Marked flatulency should be met by the prompt administration of Hoffmann's anodyne or spirit of mint. If the attacks are severe and prolonged

morphin ($\frac{1}{3}$ to $\frac{1}{4}$ grain) and atropin ($\frac{1}{120}$ grain) should be given hypodermically. If these remedies fail, recourse may be had to chloroform inhalations. The application of heat to the precordium is useful. Cardiac depression following the seizures should be combated by strychnin, ammonia, camphor, or ether.

DISEASES OF THE ARTERIES

ARTERIOSCLEROSIS

(Atheroma ; Chronic Arteritis)

Definition.—A chronic disease of the arterial walls, characterized by various degenerative changes and more or less fibrous induration.

Etiology.—It is a natural accompaniment of old age. As a presenile condition it often depends upon syphilis, gout, chronic nephritis, alcoholism, chronic lead poisoning or excessive muscular work. A not uncommon cause is long-continued high arterial tension (*hyperpiesis*), the result of over-eating, lack of physical exercise, and excessive mental strain. The disease sometimes follows acute infections, such as typhoid fever, rheumatism, etc.

Pathology.—The arteries are thickened, tortuous and rigid. The intima of the large vessels often reveals roughened and opaque areas that may be the seat of calcareous deposits. In extreme cases there may be spots of necrotic softening in the subendothelial tissue, forming so-called “atheromatous abscesses.”

Microscopic examination shows changes in all three layers. The intima is more or less thickened and often presents areas of fatty degeneration with calcification (atheromatous plaques). The media is the seat of fatty degeneration and calcification (pipe-stem arteries), or of a diffuse or patchy fibrosis with more or less calcification. The adventitia is always more or less thickened and sclerotic.

Symptoms.—These vary with extent and distribution of the sclerosis. If the process is general, it may be recognized

by rigidity and tortuosity of the accessible arteries, increasing pallor, and a gradual loss of physical and mental vigor. An increase of blood-pressure, accentuation of the aortic second sound, and signs of enlargement of the heart, especially of the left ventricle, are also commonly present, but are often absent in the senile and syphilitic forms of the disease.

If the *coronary arteries* are especially involved, the symptoms of chronic myocardial disease appear (see p. 226). If the *renal vessels* are especially affected, there may be symptoms of chronic interstitial nephritis (see p. 149). Involvement of the *cerebral arteries* may be indicated by headache, vertigo, insomnia, mental sluggishness, and, perhaps, transient paralysis. Sclerosis of the *mesenteric vessels* may lead to digestive disturbances and occasionally to attacks of abdominal pain (angina abdominis). Sclerosis of the *arteries of the limbs* may be manifested by painful muscular cramps, sudden lameness or "giving way" of the legs during walking (intermittent claudication), neuritic symptoms (numbness, tingling, darting pains, etc.), and rarely, by red, painful neuralgia (erythromelalgia).

Sequels.—The most important are cerebral hemorrhage or thrombosis, chronic myocardial disease, angina pectoris, interstitial nephritis, aneurysm, and gangrene of the extremities.

Treatment.—Treatment should be directed to the underlying condition. Alcohol should be forbidden. Tobacco should be used in great moderation. Overexertion, both mental and physical, is injurious. Gentle exercise in the open air, however, may be recommended. The quantity as well as the quality of the food demands careful revision. Turkish baths or Nauheim baths may be ordered in suitable cases. Iodids in small doses, over a long period, sometimes have a very beneficial effect. The periodic use of mild mercurial or saline aperients is serviceable. The nitrites are often useful when the blood-pressure becomes too high, but considerable caution must be exercised in their employment in nephritis as high tension is usually a conservative and com-

pensatory factor. In robust subjects moderate blood-letting sometimes affords much relief.

ANEURYSM OF THE AORTA

Definition.—A tumor-like sac, containing blood and communicating with the canal of the aorta.

Etiology.—The predisposing causes are those of arteriosclerosis—syphilis, alcoholism, gout, excessive muscular work, lead-poisoning, and nephritis. Of these, syphilis is by far the most potent factor. Immoderate physical exertion is the most common exciting cause. More than 80 per cent. of all cases occur in males. It is most frequent between the ages of thirty and fifty.

Pathology.—Aneurysms are divided, according to shape, into *fusiform*, *saccular*, and *cylindric* forms. Rupture of the intima, with the passage of blood between the outer tunics, constitutes a *dissecting aneurysm*. A *false aneurysm* is one in which all the tunics are ruptured and the extravasated blood is circumscribed by the surrounding connective tissue.

The contents of an aneurysm are either fluid or coagulated blood. In saccular aneurysms the wall is often reinforced by fibrin deposited in lamellar form.

The arch of the aorta is the most common seat. About 10 per cent. of aortic aneurysms are abdominal.

THORACIC ANEURYSM

Physical Signs.—*Inspection* may reveal a circumscribed bulging and an abnormal area of pulsation. Dilatation of the superficial veins may also be noted, and in advanced cases the skin over the prominence may be red and glossy. It is particularly important to examine the back as well as the front of the chest.

Palpation.—This may detect an expansile pulsation, a systolic thrill, and a diastolic shock from the recoil of the blood in the sac.

In aneurysm of the transverse arch a downward tug of the trachea is sometimes felt when the head is thrown back and the cricoid cartilage is grasped between the fingers and thumb (Oliver's sign).

Percussion may reveal an area of dulness to the right or left of the sternum, over the manubrium, or behind in the left inter-scapular region.

Auscultation.—Unless the sac contains too much fibrin, the ear may detect marked accentuation of the diastolic sound and a systolic murmur or bruit.

The *Roentgen Ray* is of great value in detecting aortic aneurysm and in determining the exact size and shape of the dilatation.

Pulse.—There may be inequality of the radial pulses, owing to partial blocking of a main arterial branch or to pressure on the innominate or one of the subclavian arteries by the sac itself.

Pressure Effects.—These are especially marked in aneurysms involving the transverse portion of the arch. *Dyspnea* with *stridulous inspiration* may result from pressure on the trachea or a bronchus. *Bloody sputa* ("weeping") may occur from the same cause. *Paroxysmal croupy cough* may be excited by pressure on the trachea or recurrent laryngeal nerve. *Hoarseness or aphonia* may also result from pressure on the recurrent laryngeal nerve. *Dysphagia* may result from pressure on the *esophagus*. *Pain* of a boring or lancinating character may arise from pressure on adjacent nerve-trunks or bones. Attacks of *angina pectoris* may occur as a result of the underlying aortitis. *Inequality of the pupils* and *unilateral sweating* may be excited by pressure on the sympathetic. Edema, cyanosis and enlargement of the veins of one or the other arm may arise from pressure on one of the large venous trunks.

Diagnosis.—*Mediastinal tumor* may simulate aneurysm, but in the former the pulsation is not expansile, there is no diastolic shock, the tracheal tug is usually absent, and there may be cachexia, enlargement of superficial glands, and leukocytosis.

Pulsating Empyema.—A left-sided purulent effusion may transmit a cardiac pulsation, but there is no diastolic shock, no thrill, and no murmur. The history, moreover, will usually suggest pleurisy.

Aortic Stenosis.—In this condition there are no evidences of a tumor, no pressure symptoms, and no inequality in the radial pulses.

Prognosis.—The outlook is grave. Death usually occurs in from one to three years from rupture, asphyxia, exhaustion, septic inflammation of the lung ("aneurysmal phthisis"), or cerebral embolism. Rupture may take place into the trachea, a bronchus, the pleura, lung, pericardium or esophagus. Occasionally recovery follows from clot-formation.

Treatment.—The treatment commonly employed is a modification of Tufnell's method, and consists in absolute rest in bed for a period of six or eight weeks, a comparatively dry diet, and the administration of potassium iodid (20 to 30 grains thrice daily). In recent syphilitic cases, if there are no signs of myocardial insufficiency, arsphenamin and mercury may be used with hope of destroying the spirochetes in the vascular tissue. For severe pain the most effective measures are the application of an ice-bag and the administration of nitroglycerin or morphin. When there is marked dyspnea with cyanosis, venesection may afford relief. In some instances the introduction of gold wire into the sac and the passage of galvanic current through the wire (Moore-Corradi method) give excellent results. The operation is not a dangerous one and often relieves pain and prolongs life. It is only suitable for saccular aneurysms.

ANEURYSM OF THE ABDOMINAL AORTA

Seat.—It is most frequently located near the celiac axis.

Symptoms.—It may be recognized by sharp pain in the back, radiating along the spinal nerves, by retardation and weakness of the femoral pulse, by gastro-intestinal symptoms, and by physical signs similar to those of thoracic aneurysm.

Diagnosis.—An *abdominal tumor* may receive a pulsation from the aorta and simulate aneurysm, but in the former the pulsation is not expansile, and is frequently lost when the patient is placed in the knee-breast posture.

Expansile Abdominal Aorta.—This is most frequently seen in women, in whom abdominal aneurysm is rare; the pulsation is often paroxysmal; there is no distinct tumor, and there are no pressure symptoms.

Course and Treatment.—Death usually results from rupture. Occasionally the fatal issue is effected through erosion of the vertebræ and paraplegia, or through embolism of the superior mesenteric artery. The treatment is that of thoracic aneurysm.

THROMBO-ANGIITIS OBLITERANS

This is a progressive inflammatory condition of the arteries and veins, chiefly those of the legs, resulting in thrombosis, organization of the clot, and conversion of the affected vessel into a fibrous cord. The disease, which is comparatively rare, occurs chiefly in males between the ages of twenty and forty and is confined almost entirely to Russian Jews. The chief *symptoms* are pain in the affected member, especially when it hangs down, intermittent claudication, absence of the pulses, migrating phlebitis of certain superficial veins, and eventually gangrene. *Treatment* is rarely more than palliative. Applications of superheated air, passive hyperemia by Bier's method, intravenous injections of a solution of sodium citrate (2 per cent.), and the administration of potassium iodid by the mouth are the most promising measures.

DISEASES OF THE RESPIRATORY SYSTEM

GENERAL SYMPTOMATOLOGY

THE NOSE

The Sense of Smell.—This is tested by holding odorous substances before one nostril at a time while the other is closed. Pungent vapors should be avoided, as the irritation which they excite, and not their odor, may lead to their recognition.

The sense of smell may be impaired or lost (anosmia) in:

(1) Lesions of the brain affecting the olfactory tract or bulb (contusions, tumor, meningitis); (2) lesions of the olfactory nerves (rhinitis, neuritis from influenza, etc.); (3) hysteria; (4) paralysis of the trigeminal nerve, causing dryness and trophic changes in the Schneiderian membrane:

Extreme acuteness of the sense of smell (hyperosmia) and perversions of the sense of smell (parosmia) are sometimes observed in hysteria, epileptic aura, and organic disease of the brain.

Epistaxis.—Hemorrhage from the nose occurs under the following conditions: (1) Traumatism; (2) inflammation or ulceration; (3) new growths; (4) high blood-pressure, as in arteriosclerosis, chronic glomerulonephritis, and hypertrophy of the heart; (5) venous stasis from chronic heart or liver disease; (6) blood dyscrasias, as in hemophilia, purpura, scurvy, pernicious anemia, etc.; (7) onset of acute infections, especially typhoid; (8) ascent to high altitudes; (9) vicarious menstruation (rare).

THE LARYNX

Spasm of the laryngeal adductors is characterized by intense dyspnea and occurs in spasmodic croup; in true croup; in ulceration of the larynx; in laryngismus stridulus; in whooping-cough; in tetany; in hysteria; in hydrophobia; in the laryngeal crises of locomotor ataxia; when foreign bodies have lodged in the larynx; and when aneurysms or mediastinal tumors press on the recurrent laryngeal nerve and irritate it.

Aphonia or loss of voice may be due to: (1) Organic disease of the larynx—inflammation, neoplasms, cicatricial stenosis. (2) Centric paralysis of the recurrent laryngeal nerves, as in bulbar palsy. (3) Peripheral paralysis of the recurrent laryngeal nerves caused by pressure of an aneurysm, mediastinal tumor, or pericardial effusion. (4) Hysteria. (5) The lodgement of foreign bodies. (6) Prolonged use of the voice.

RESPIRATION

Dyspnea.—Dyspnea implies difficult breathing, with or without an increase in the number of respirations. Dyspnea which is so severe as to necessitate a sitting posture is termed orthopnea. Dyspnea may occur on inspiration, expiration, or both.

Its *chief causes* are: (1) Obstruction in the larynx from spasm, paralysis, false membrane, edema, or a foreign body. (2) Pressure of an aneurysm, a tumor, or large glands upon the trachea, a bronchus, or the recurrent laryngeal nerve. (3) Asthma. (4) Diseases of the lungs, as pneumonia, emphysema, edema, etc. (5) Pleural effusions. (6) Cardiac disease. (7) Paralysis of the muscles of respiration. (8) Abdominal distention. (9) Anemia.

Inspiratory dyspnea is especially marked when there is obstruction in the upper air passages—larynx or trachea.

Expiratory dyspnea is noted in emphysema and occasionally in movable tumors situated below the glottis. In asthma, also, the dyspnea may be largely expiratory.

The Number of Respirations a Minute.—In the healthy male adult the number of respirations is about 18 a minute. In women and children breathing is somewhat more rapid. The ratio between respirations and pulse-beats is as 1 is to 4 or 4.5.

Rapid respiration (polypnea) is noted in excitement; in pyrexia; in inflammatory diseases of the lungs; in anemia; in certain affections involving the base of the brain; in poisoning from certain drugs that affect the respiratory center; in hysteria; in painful affections of the chest, as pleurodynia and pleurisy.

Infrequent respiration is observed in certain diseases of the brain, as meningitis, tumor, apoplexy; in advanced fatty degeneration of the heart; in certain forms of coma, particularly uremic and diabetic; in poisoning with certain drugs, especially opium; in obstruction to the air-passages, as in asthma and in laryngeal spasm.

Cheyne-Stokes, or Tidal-wave Breathing.—In this type the respirations gradually increase in depth until they reach a climax, then gradually subside, and finally cease entirely for from five to fifty seconds, when they begin again. Consciousness is sometimes lost during the pause. Cheyne-Stokes breathing depends on some disturbance of the respiratory center the exact nature of which is still undetermined. It is usually a forerunner of death, but in some cases of cardiac and renal disease it persists for months.

Its *chief causes* are: (1) Certain cerebral diseases, as apoplexy, meningitis, and tumor. (2) Advanced cardiac disease, especially fatty or fibroid heart. (3) Certain forms of coma, especially that produced by uremia, opium-poisoning, and sunstroke.

COUGH

Cough may be induced by: (1) Most organic diseases of the pharynx, larynx, bronchi, and lungs. (2) Foreign bodies in the air-passages. (3) Certain infections which are asso-

ciated with catarrh, such as typhoid fever, measles, whooping-cough, and influenza. (4) Inhalation of irritant dusts or vapors. (5) Irritation of nerves that are in anatomic relation with the vagus. (6) Hysteria.

Laryngeal Cough.—This cough has a hard, metallic, ringing intonation, and has been termed “croupy.” It is observed in laryngitis; in whooping-cough; in tuberculosis and syphilis of the larynx; when a foreign body is lodged in the larynx; when the recurrent laryngeal nerve is irritated by pressure of a tumor or aneurysm; and in hysteria.

Dry Cough.—Cough without expectoration is especially observed in the beginning of inflammatory diseases of the bronchi and lungs; in pleurisy; in most chest diseases of early childhood; and in reflex irritation of the larynx.

Moist or loose cough occurs especially in bronchitis, bronchiectasis, pulmonary edema, pulmonary tuberculosis, in pneumonia after the crisis, and abscess of the lung.

EXPECTORATION

Mucoid sputum is noted especially in the beginning of acute bronchitis; in asthma; in the early stages of pneumonia and tuberculosis; and in pulmonary edema. In edema the sputum is very frothy and watery.

Mucopurulent sputum is observed in acute bronchitis (later stages), chronic bronchitis, bronchiectasis, pneumonia (after the crisis), pulmonary tuberculosis (later stages), pulmonary abscess, and empyema communicating with the lung. In the last two conditions the expectoration may be composed of pure pus.

Hemorrhagic Sputum.—Sputum has a reddish, brownish, or prune-juice color when mixed with blood. In some cases the sputum consists of pure blood (see hemoptysis). Hemorrhagic expectoration occurs in tuberculosis of the lungs, in pneumonia (especially croupous pneumonia), in hemorrhagic infarctions of the lungs, in abscess and gangrene of the lungs, in tumors of the lungs, and, finally, in congestion of the pul-

monary circulation. In cancer of the lungs the sputum sometimes resembles red-currant jelly, and in hepatopulmonary abscesses (amebic) the expectoration has been likened to anchovy sauce.

Albuminous Sputum.—The expectoration of large quantities of serous albuminous fluid rarely occurs suddenly after the aspiration of pleuritic effusions. It is sometimes followed by fatal edema of the lungs.

Fetid sputum usually results from bronchiectasis, advanced pulmonary tuberculosis with cavities, gangrene of the lung, or abscess of the lung.

Such sputum, if allowed to stand in a conic glass, settles in three layers: an upper layer of discolored froth, a middle layer of turbid mucus in which are suspended purulent strings, and a bottom layer of decomposed pus.

Nummular sputum is sputum occurring in round, flat, coin-shaped masses, which sink in water. Expectoration of this character is seen in advanced pulmonary tuberculosis and occasionally in bronchiectasis.

The Microscopy of Sputum.—*Elastic fibers* are found in the sputum in pulmonary tuberculosis, abscess, gangrene of the lungs, and in some cases of bronchiectasis.

The Detection of Elastic Fibers.—Place the sputum which has collected during the night in a glass beaker, and add to it an equal volume of a solution of caustic soda (20 grains to the ounce), and boil over a spirit-lamp, stirring it occasionally with a glass rod. As soon as it boils, pour into a conic glass, and add four or five times the amount of cold distilled water. Allow the mixture to stand for two to three hours, and examine the sediment as for tube casts (Fenwick).

Spirals of Mucin.—Tightly coiled spirals of mucin, which probably represent molds of the fine bronchioles, were first pointed out by Curschmann in the sputum of asthma. They have also been observed in the sputum of croupous pneumonia.

Charcot-Leyden Crystals.—These are small transparent octahedral crystals, similar to those found in the blood in leukemia.

They are observed especially in the sputum of asthma. They have also been noted in tuberculosis, in fibrinous bronchitis, and in acute bronchitis.

Crystals of Fatty Acids.—These occur as fine needles, singly or in bundles, and are often sharply curved near their extremities. They are observed in the sputum of chronic bronchitis, of abscess, and of gangrene of the lungs.

Crystals of Hematoidin.—These occur as small yellow needles, rhombic plates or tufts, and are found in sputa which contain altered blood. They may be observed in abscess, gangrene, and cancer of the lungs.

PHYSICAL EXAMINATION OF THE RESPIRATORY ORGANS

Inspection.—Inspection reveals the shape of the chest, any unnatural prominence or depression, the amount of expansion, and any inequality of expansion.

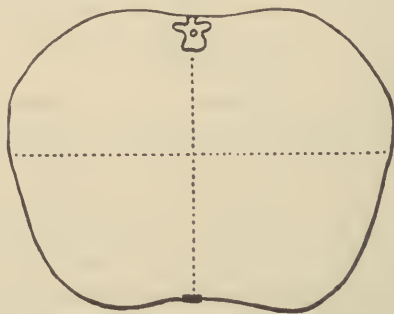


FIG. 8.—An outline of the normal chest.

Phthisinoid Chest.—The anteroposterior diameter is short; the thorax is long and flat; the ribs are oblique; the scapulæ are prominent; the spaces above and below the clavicles are depressed; and the angle formed by the divergence of the costal margins from the sternum is very acute.

Rachitic Chest.—This may resemble the former, but usually the sides are considerably flattened and the sternum prominent, so that the term “pigeon-breast” has been applied to

this particular form. The sternal ends of the ribs are enlarged or "beaded," and this characteristic has given rise to the term "rachitic rosary." There is often a circular constriction of the thorax at the level of the xiphoid cartilage (Harrison's groove).

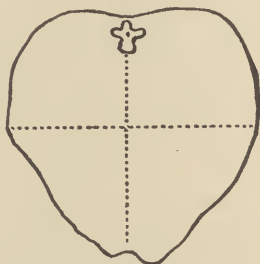


FIG. 9.—Rachitic chest.

Emphysematous Chest.—In advanced emphysema the thorax is short and round; the anteroposterior diameter is often as long as the transverse; the ribs are horizontal; and the angle formed by the divergence of the costal margins from the

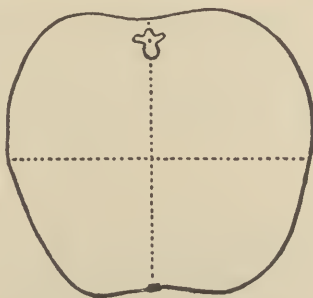


FIG. 10.—Emphysematous chest.

sternum is very obtuse. The term "barrel-shaped chest" is applied to this configuration.

Local Prominences and Depressions.—An unnatural prominence or depression is often observed over the lower part of the sternum, and is usually congenital. The term "funnel

breast" or "shoemaker's breast" (because it may result from the pressure of tools) has been applied to the sternal depression.

A unilateral or local depression may be due to: (1) Chronic pulmonary tuberculosis; (2) cirrhosis of the lung; (3) pleurisy with fibrous adhesions.

A unilateral or local prominence may be due to: (1) Pleurisy with effusion; (2) pneumothorax, hydrothorax, hemothorax; (3) an aneurysm or tumor; (4) compensatory emphysema, resulting from impairment of the opposite lung; (5) cardiac enlargements (left side); (6) enlargements of the abdominal organs, especially the liver and spleen.

Expansion.—In women and in children, and in both sexes during sleep, breathing is largely thoracic or costal; in men and in the aged of both sexes it is largely abdominal or diaphragmatic.

Restricted abdominal breathing is observed in pregnancy; in abdominal tumors and effusions; in peritonitis; in diaphragmatic pleurisy; in paralysis of the phrenic nerve from pressure or from bulbar disease; and occasionally in the "hysteric abdomen."

Diminished expansion of one side is observed in pleural effusions; in acute pleurisy (from pain); in consolidation of the lung from tuberculosis, pneumonia, or tumor; in occlusion of a bronchus; and in marked enlargement of the liver or spleen.

Increased expansion of one side is observed in compensatory emphysema.

Litten's Diaphragm Phenomenon.—If a healthy individual is placed in a horizontal position with the feet toward the window and all cross-lights are excluded, a narrow shadow may be seen descending between the sixth and the ninth ribs in each axilla during full inspiration. It is due to the separation of the diaphragmatic pleura from the costal pleura during the inspiratory descent of the diaphragm. This shadow is absent in pleural effusion, pneumonia of the lower lobe, well-developed emphysema, and extensive pleuritic adhesions. As

it is present in enlargement of the liver and in subphrenic abscess, it often aids materially in determining whether disease is above or below the diaphragm.

Palpation.—Palpation serves to detect any thoracic tenderness, edema, friction fremitus, or râles, and to determine the vocal fremitus and amount of expansion.

Thoracic tenderness is observed in pleurisy; in pulmonary tuberculosis and pneumonia (from associated pleurisy); in pleurodynia; in intercostal neuralgia (confined to certain spots); in caries and fracture of the ribs; and in contusion and inflammation of the parietes.

Edema of the chest-walls is recognized by “pitting” when pressure is made with the finger. It may be observed in empyema; in deep-seated abscesses of the parietes; after the application of a blister; and in general dropsy.

Friction Fremitus and Râles.—The friction-rub of pleurisy and harsh, sonorous râles can sometimes be detected by palpation.

Vocal fremitus (tactile fremitus) is the purring vibration imparted by the voice to the palpating hand.

In determining the vocal fremitus observe the following precautions: Palpate symmetric parts of the chest; make firm pressure; when comparing, use the same pressure on the two sides; apply the hands as nearly parallel to the ribs as possible; and bear in mind that the fremitus is normally stronger over the right chest than the left.

The fremitus is usually light in women and in children, and in men with thick chest-walls and a weak voice.

Vocal fremitus is abnormally increased in all consolidations of the lung with unobstructed bronchi. It is usually increased in—(1) Tuberculosis; (2) croupous pneumonia; (3) bronchopneumonia. It may be decreased in any of these conditions, however, if the bronchioles contain exudation.

Vocal fremitus is decreased or absent in—(1) Pleural effusions and pneumothorax; (2) emphysema; (3) pulmonary collapse from an obstructed bronchus; (4) pulmonary edema; (5) morbid growth of the lung (if the bronchi are compressed).

Percussion.—Percussion, through the sounds and resistance elicited, serves to determine the boundaries and physical condition of the underlying organs.

Immediate percussion is performed by striking the chest directly with the fingers. It is not often employed, except over the clavicles, where the bones themselves act as pleximeters.

Mediate percussion is performed by using the fingers of one hand as a plexor and those of the opposite hand as a pleximeter; or by using a piece of ivory, glass, or hard rubber as a pleximeter, and a small hammer as a plexor.

The use of the fingers alone is preferable, for only in this way can resistance be determined.

In percussion the following precautions should be observed: Place the finger that is being used as a pleximeter firmly against the chest, and preferably parallel to the ribs; make the finger that is used as plexor strike the one on the chest perpendicularly; fix the forearm, and use no more force than can be obtained from a gentle swing of the wrist. When possible, percuss all parts of the chest anteriorly and posteriorly; percuss both in inspiration and in expiration. In comparing the two sides, be sure to percuss symmetric parts.

Normal Resonance.—On the right side, pulmonary resonance extends from a half inch to an inch above the clavicle, downward to the upper border of the sixth rib in front, and to a line drawn through the tenth spinous process posteriorly.

On the left side, pulmonary resonance extends from a half inch to an inch above the clavicle, downward, within the midclavicular line to the third rib, outside of the midclavicular line to the tenth rib, and posteriorly to a line drawn through the tenth spinous process.

Traube's Semilunar Space.—This is a tympanitic area at the base of the left chest, bounded above by the lung (sixth rib), on the right by the liver, and on the left by the spleen. It is obliterated in pleural effusion on the left side.

Hyperresonance is observed in the following conditions: (1) Pneumothorax. (2) Cavities—tuberculous or bronchiec-

tatic. (3) Emphysema. (4) Lowered pulmonary tension, as above a pleural effusion or consolidation, and in the initial state of pneumonia (Skoda's resonance). (5) Flatulent distention of the stomach or colon (frequently observed over the base of the left chest).

Tympanitic resonance is resonance of a hollow, drum-like character, like that normally obtained by percussing the empty stomach or the colon. It is elicited over the chest in pneumothorax and cavity-formation.

The cracked-pot sound, or *bruit de pôt fêlé*, is a modified tympanitic sound, and can be simulated by percussing over the cheek when the mouth is partially open. It may be normally heard over the chest of a crying infant (Walshe). In the adult it usually indicates a cavity that has a free communication with a bronchus. It is best detected by keeping the ear near the open mouth of the patient while percussing.

Dulness or flatness on percussion may be caused by the following conditions: (1) Pleurisy with effusion; (2) consolidation of the lung from tuberculosis or pneumonia; (3) collapse of the lung; (4) congestion and edema of the lung; (5) morbid growths in the lung; (6) enlargement of the liver or spleen (at the bases).

Pitch.—Pitch of the note depends largely upon the volume of air, upon the tension of the walls of the cavity, and upon the size of the opening that communicates with the cavity. The less the air, the greater the tension, and the smaller the opening, the higher will be the pitch of the note. It is obvious, therefore, that conditions that are associated with hyperresonance may yield either a high- or a low-pitched note. In beginning tuberculous consolidation the note over the affected apex is higher pitched; but it must be borne in mind that normally the note over the right apex is higher pitched than that over the left.

Resistance.—The sense of resistance appreciated by the percussing finger is increased in proportion as the air in the lungs is decreased. It is generally more marked over

a pleural effusion than over a consolidation with patulous bronchi.

Auscultation.—Auscultation of the lungs is practised to determine the character of the respiratory and voice sounds, and to detect adventitious sounds, as râles.

In *immediate auscultation* the ear is placed directly over the chest, only a soft towel intervening.

In *mediate auscultation* the sounds are transmitted through a stethoscope, which should be applied to the bare chest.

In auscultation observe the following precautions: Do not exert much pressure with the stethoscope. If the chest is covered with hair, this should be moistened, otherwise it is likely to produce crackling sounds resembling râles. If possible, examine carefully all parts of the chest, anteriorly and posteriorly, during quiet breathing, during full inspiration, during full expiration, and after coughing. Compare carefully the sounds elicited over symmetric parts of the chest.

Normal Respiration.—Vesicular breathing is heard over the body of the lungs and is characterized by a soft, breezy inspiratory sound, and a shorter, lower pitched, less intense expiratory sound. Normally, the expiratory sound is not more than one-third the length of the inspiratory sound. Not infrequently expiration is wholly inaudible. Over the trachea and main bronchi the sounds are harsh and blowing, and the expiratory sound is as long as the inspiratory sound or slightly longer (bronchial breathing).

Modifications of the Respiratory Murmur.—*Puerile Breathing and Exaggerated Breathing.*—Normal breathing in children is called puerile breathing. Both inspiration and expiration are proportionately increased in length and loudness. Exaggerated breathing has the same characteristics as puerile breathing, and is heard after exertion and over the whole of one lung (compensatory emphysema) if the other is disabled.

Bronchial or Tubular Breathing.—This is harsh, blowing breathing with prolonged, accentuated expiration. The expiration may be considerably longer than the inspiration,

and there is often a distinct pause between the two sounds. Bronchial breathing is heard normally over the trachea and in the interscapular space over the large bronchi, and abnormally over consolidated lung, if the bronchi are unobstructed. Thus, it may be heard in croupous pneumonia, in bronchopneumonia, in tuberculosis of the lung, and over the lung above a pleural effusion.

Bronchovesicular Breathing.—This is breathing that is neither bronchial nor vesicular. It is somewhat harsh, and expiration is slightly prolonged and accentuated. Unlike puerile or exaggerated breathing, the normal ratio of inspiration to expiration is not maintained—the expiration is not relatively but *actually* prolonged. Bronchovesicular breathing may be heard when the lung is slightly solidified, as in beginning tuberculosis.

Cavernous or Amphoric Breathing.—This resembles bronchial breathing, but the sounds have a hollow character, and the pitch of the expiration is lower than that of inspiration. It may be imitated by blowing over the mouth of an empty jar.

Cavernous breathing may be heard in the following conditions: (1) Tuberculous or bronchiectatic cavities; (2) pneumothorax, if the opening in the lung is patulous; (3) areas of consolidation near a large bronchus; (4) sometimes over lung compressed by a moderate effusion.

The Breathing of Emphysema.—This is weak breathing, with prolonged low-pitched or inaudible expiration.

Cogged-wheel or Jerky Breathing.—The respiratory murmur is not continuous, but is broken into waves. It is not indicative of any special disease but it is frequently observed in hysteria, pleurodynia, bronchitis, and incipient tuberculosis.

Weak or Shallow Breathing.—This is noted: (1) When the chest-walls are thick; (2) in the old and feeble; (3) in emphysema; (4) in pleural effusion; (5) sometimes in incipient tuberculosis; (6) in painful affections of the chest, as pleurodynia and beginning pleurisy; (7) in pulmonary edema.

Vocal Resonance.—This is the confused humming sound heard over the chest when the patient speaks. It is modified by the same conditions that modify the vocal fremitus (see page 245).

Bronchophony.—This is exaggerated vocal resonance. It is heard normally over the trachea, and abnormally over consolidated lung (tuberculosis and pneumonia) if the bronchi are unobstructed, over lung that is compressed by pleural effusion, and over some cavities.

Pectoriloquy.—This is a modification of vocal resonance in which the articulate speech is heard very distinctly, as though coming directly from the chest into the ear. It is more pronounced when the patient whispers.

Pectoriloquy is heard over: (1) Cavities that communicate with a bronchus; (2) areas of consolidation in the neighborhood of a large bronchus; (3) pneumothorax, if the opening in the lung is patulous; (4) some pleural effusions.

Egophony.—This is a modification of bronchophony, in which the sounds have a trembling or bleating quality. It is usually heard over slight pleural effusions near the upper border of dulness, especially near the inferior angle of the scapula.

Adventitious Sounds.—These are not modifications of pre-existing sounds, but wholly new sounds produced in the lung or pleura. They include râles, friction-sounds, metallic tinkling, and succussion-splash.

Râles.—These are new sounds created in the trachea, bronchi, air-vesicles, or in cavities. They may be due to the passage of air through secretions or other fluid or to the separation of adherent alveolar walls by the inspiratory air.

Pulmonary râles	Bronchial	Dry	<ul style="list-style-type: none"> { Sibilant { Sonorous
		Moist	<ul style="list-style-type: none"> { Subcrepitant { Bubbling { Gurgling
	Vesicular = Crepitant		

Dry râles are probably produced by the passage of air over threads of viscid secretion drawn across the bronchial lumen. They have a musical character and are often transmitted to a considerable distance from the point of production. They are heard particularly in bronchitis and asthma. Sibilant râles are whistling and high pitched; sonorous râles have a humming quality and are lower pitched. Dry râles may be heard on inspiration, expiration, or on both.

Moist râles are probably produced by the rupture of films or membranes of thin secretion stretched across the bronchial lumen. The thinner the liquid and the larger the tube, the coarser will be the râles. They may be heard on inspiration, expiration, or on both.

Subcrepitant or *crackling râles* are fine moist râles. They are heard in all conditions that are associated with liquid in the smaller tubes, such as bronchitis, bronchopneumonia, pulmonary edema, and beginning tuberculosis.

Bubbling râles are coarser than subcrepitant, and are heard in bronchitis, in resolving croupous pneumonia, over softening tuberculous deposits, and over small cavities.

Gurgling râles are very coarse, and resemble the bursting of large bubbles. They are heard over large cavities that contain fluid, and over the trachea in the so-called "death-rattle."

Crepitant Râles.—These are very fine râles, usually heard at the end of full inspiration. They may be simulated by rubbing a lock of hair between the fingers. They have been especially associated with the first stage of croupous pneumonia, and have been ascribed to the forcible separation of adherent vesicular walls. Râles very similar to, if not identical with, these are heard in pulmonary edema.

Friction-sounds are produced by the rubbing together of roughened pleural surfaces. They may be heard both in inspiration and in expiration, and often resemble subcrepitant râles, but they are more superficial and localized than the latter, and are not modified by cough or deep inspiration.

A roughened pleura in the neighborhood of the heart may produce a friction-sound of cardiac rhythm, and one which will still continue when the breath is held; under other conditions pleural friction-sounds cease when respiration is suspended.

Metallic Tinkling.—This name is applied to silvery or bell-like sounds that are heard at intervals over a pneumo-hydrothorax or large cavity. Speaking, coughing, and deep breathing usually induce them. Care must be taken not to confound them with similar sounds produced by the presence of liquid in a distended stomach.

Succussion-splash or Hippocratic Succussion.—This is a splashing sound produced by the presence of air and liquid in the chest. It may be elicited by gently shaking the patient while auscultating. It is usually associated with hydro-pneumothorax or a pyopneumothorax.

A similar splashing sound is often heard over a dilated stomach.

Mensuration.—In measuring the sides of the chest observe the following precautions: Measure from the middle of the sternum to the spinous processes; measure both sides after inspiration and after expiration; apply the tape with equal firmness to the two sides. In comparing, measure corresponding levels, and remember that the right side is from half an inch to an inch greater in circumference than the left.

The conditions that render one side more prominent than the other have already been considered.

Roentgen Ray.—In certain pathologic conditions within the chest the x-ray furnishes valuable information. It has been found especially useful in detecting aortic aneurysms, in determining the outline of the heart in emphysema, in recognizing pulmonary tumors and tuberculous infiltration in doubtful cases, and in locating small pleural effusions.

DISEASES OF THE NOSE AND LARYNX

ACUTE CORYZA

(Acute Rhinitis ; Cold in the Head)

Acute coryza is an infection of the nasal cavities, sometimes extending into the pharynx, upper respiratory tract, Eustachian tubes and accessory nasal sinuses. Exposure to cold and wet may act as a predisposing factor, but the exciting cause is microörganismal. In some cases coryza is symptomatic of a general infection, such as measles or influenza, of a drug intoxication, such as iodism, or of protein sensitization (hay fever), or results from the inhalation of irritant dust or vapors.

Symptoms.—The disease is ushered in with chilliness, muscular soreness, general malaise, fulness in the head, and sneezing. The nasal chambers are obstructed, so that the patient is obliged to breathe through his mouth. At first there is no secretion, but in twenty-four or forty-eight hours a watery discharge is established, which later becomes mucopurulent. Slight fever and its associated symptoms are commonly present. The duration is from a few days to two weeks.

Complications.—Extensions of the disease to the accessory nasal sinuses, Eustachian tube, middle ear, pharynx, larynx, and bronchi are not uncommon. Repeated attacks may lead to chronic rhinitis.

Treatment.—If the patient is seen at the outset and is willing to remain indoors for twenty-four hours, a hot foot-

bath, with a full dose of Dover's powder, followed in the morning by a Seidlitz powder or other saline aperient, often gives excellent results. When the patient must be up and about, the following capsules will usually afford considerable relief:

R̄.	Codeinæ sulphatis.....	gr. iiss
	Pulveris camphoræ.....	gr. xx
	Ammonii carbonatis.....	ʒj
	Quininæ bisulphatis.....	gr. xx
	Extracti belladonnæ.....	gr. j.—M.

Pone in capsulas No. xx.

SIG.—One every two or three hours.

Warm Dobell's solution (see p. 37) or warm distilled extract of witch-hazel (diluted with 1 part of water) used as a spray at intervals, and followed in a few minutes by an oily application, such as the following, usually renders satisfactory service:

R̄.	Mentholis.....	gr. iiij
	Petrolati liquidi.....	q. s. ad fʒj.—M.

CHRONIC NASAL CATARRH

(Chronic Rhinitis)

Definition.—A chronic inflammation of the nasal mucous membrane.

Etiology.—Repeated attacks of acute coryza, impure air, the continual inhalation of irritating dusts or vapors, lowered vitality, and congenital or acquired obstruction of the nasal chambers are causal factors. It is sometimes an expression of syphilis.

Varieties.—(1) Simple chronic rhinitis; (2) hypertrophic rhinitis; (3) atrophic rhinitis.

Symptoms.—These consist of a mucoid or mucopurulent discharge from the nose; obstruction of the nostrils from swelling or hypertrophy of the mucosa or from inspissated secretion; mouth-breathing; a nasal intonation of the voice; frontal headache; and impairment of the sense of smell.

Symptoms of catarrh of the adjacent organs are frequently present. The most common of these are: dryness of the throat and hawking from pharyngitis; deafness from catarrh of the middle ear; and watering of the eyes from catarrhal occlusion of the lacrimal canal.

Simple Chronic Rhinitis.—The mucous membrane of the nose is congested, swollen, and highly irritable. There is hypersecretion of mucus or mucopus.

Hypertrophic Rhinitis.—The mucous membrane is red and the cavities are more or less occluded from hypertrophy of the cavernous tissue covering the turbinated bones. In advanced cases exostoses from the bony framework are sometimes noted. The secretion is usually composed of thick mucopus. Adenoid growths are often found in the nasopharynx.

Atrophic Rhinitis (Ozena).—This form is seen most frequently in young adults, and is more common in women than in men. The nasal chambers are large; the mucous membrane is pale, dry, and glazed; adherent scabs are usually present. The secretion is very abundant, thick, and of a yellowish or greenish color. A characteristic feature is the extremely offensive odor, which is probably due to the decomposition of the retained secretion. In advanced cases there may be necrosis of the bones and sinking in of the bridge of the nose.

Prognosis.—In the simple and hypertrophic form the prognosis is favorable under persistent treatment. In atrophic rhinitis perfect cure is rarely attainable, but great improvement is possible.

Treatment.—Any constitutional vice, if present, should receive appropriate treatment. Fresh air, outdoor exercise, and frequent bathing, with friction of the skin, are to be recommended. Tonics, especially strychnin and cod-liver oil, are often required. The nasopharynx must be kept clean by frequent spraying with an antiseptic alkaline liquid, such as Dobell's solution (see p. 37).

In the hypertrophic form local remedies of an astringent or alterative character are often efficacious. The following are in common use: A mixture of iodine and glycerin containing 6 grains of iodine, 12 grains of potassium iodide, and 1 ounce each of glycerin and water; aqueous solution of ichthyol (20 to 40 per cent.); solution of zinc sulphocarbolate (2 to 5 per cent.); and solution of silver nitrate (1 to 2 per cent.).

If the hypertrophic process prove resistant, the obstruction must be removed by means of caustics (chromic or trichloroacetic acid), the galvanocautery, or the snare.

In atrophic rhinitis the crusts may be removed by pledges of cotton soaked in a solution of hydrogen dioxide. After the nares have been thoroughly cleansed, an oily solution like the following may be applied:

R̄.	Mentholis.....	gr. xx
	Thymolis.....	gr. vj
	Eucalyptolis.....	℥xx
	Petrolati liquidi.....	f℥vj.—M.

For destroying the offensive odor one of the following applications may be used: Instillations of ichthyol (15 to 50 per cent. solution every other day), or sprays of formaldehyd (1 to 5000) of potassium permanganate (2 grains to the ounce).

ACUTE CATARRHAL LARYNGITIS

Definition.—An acute catarrhal inflammation of the larynx.

Etiology.—Faulty use of the voice, exposure to cold and wet, the inhalation of irritating dusts or vapors, the use of certain drugs (iodides, mercury), and the lodgment of foreign bodies may cause it. It is also an associated condition in certain infectious diseases, like whooping-cough, measles, diphtheria, and influenza. The organisms most commonly found are the *Micrococcus catarrhalis*, *pneumococcus*, *influenza bacillus* and *streptococcus*.

Symptoms.—The chief symptoms are: Hoarseness of the voice or aphonia; hard, ringing cough; pain in the throat, increased by speaking, coughing, and swallowing; expectoration, which is at first scanty and later mucopurulent; slight fever and its associated symptoms. In sensitive persons, and especially in children, paroxysms of croupy cough and dyspnea (*false croup*) may result from spasm of the vocal cords. When there is much edema, severe dyspnea becomes a prominent feature.

Inspection.—The mucous membrane of the laryngeal walls and vocal cords is red and swollen. In grave cases the tissues are highly edematous.

Prognosis.—In simple laryngitis without edema the prognosis is altogether favorable. The attack usually lasts from a week to ten days. When there is edema of the larynx, indicated by dyspnea or threatened asphyxia, the prognosis is grave.

Treatment.—Use of the voice should be avoided. The air of the room should be rendered moist by means of steam. An ice-bag or iodine may be applied externally. At the onset it is advisable to administer a mild aperient. Mild expectorants—ipecac, potassium citrate or ammonium chlorid—may be given in conjunction with heroin, codein, or paregoric if the cough is troublesome. Such a combination as the following is often serviceable:

R̄.	Potassii citratis.....	℥iij
	Vini ipecacuanhæ.....	f℥iv
	Tincturæ opii camphoratæ.....	f℥iv
	Syrupi tolutani.....	f℥iss
	Aquæ.....	q. s. f℥iv.—M.

SIG.—A dessertspoonful every three hours.

In acute *edematous laryngitis*, if the swelling does not yield to local bloodletting, the external application of ice, astringent sprays (solution of adrenalin chlorid), and scarification of the mucous membrane, and the symptoms are urgent, tracheotomy should be performed without delay.

SPASMODIC CROUP

(False Croup; Catarrhal Croup)

Definition.—Spasm of the vocal cords, excited by catarrh of the larynx.

Etiology.—The attacks usually occur in young children, and are induced by the causes of catarrhal laryngitis.

Symptoms.—Usually there has been a little hoarseness and cough during the day, and at night the child is awakened from sleep by a severe paroxysm of suffocative cough. The latter has a peculiar hard, metallic quality, and is associated with the evidences of dyspnea, namely, anxious face, dilating nostrils, prominent sternocleidomastoids, and retraction of the base of the chest with each inspiratory effort. During the paroxysm the skin is hot and the pulse is tense and rapid. In from a few moments to an hour the cough ceases, free perspiration follows, and the child falls asleep.

Two or three similar attacks may occur in the same night but on the following day the child may appear quite well. A recurrence of the seizures for several successive nights is not infrequent.

Diagnosis.—*Membranous or Pseudomembranous Croup (Diphtheria).*—Hoarseness and dyspnea develop gradually, and the latter is not intermittent. False membrane may be seen in the throat or may be coughed up. The constitutional symptoms are more severe.

Laryngismus Stridulus.—This is a pure neurosis, and is often associated with rickets. The paroxysms resemble those of false croup, but are associated with a peculiar crowing inspiration, and lack catarrhal symptoms, such as hoarseness and cough.

Prognosis.—The prognosis is good.

Treatment.—A sponge moistened with hot water may be applied to the throat, or the child may be placed in a hot bath. If these simple measures fail, an emetic will almost invariably bring relief. Wine of ipecac (1 dram) may be

selected. Subsequent treatment should be directed to the laryngeal catarrh. A mixture like the following will be found useful:

R̄.	Tincturæ aconiti.....	℥xx
	Vini ipecacuanhæ.....	f℥j
	Potassii bromidi.....	℥ss
	Potassii citratis.....	℥j
	Syrupi tolutani.....	f℥j
	Aquæ.....	q. s. ad f℥ij.—M.

SIG.—A teaspoonful every two of three hours for a child of two years.

MEMBRANOUS CROUP

(Croupous Laryngitis; True Croup; Pseudomembranous Laryngitis)

See Laryngeal Diphtheria (p. 356).

CHRONIC LARYNGITIS

Simple Chronic Catarrhal Laryngitis.—This may follow an acute attack, or it may develop gradually from overuse of the voice, excessive smoking, or inhalation of dust or irritant vapors.

Symptoms.—These consist in moderate hoarseness, aphonia after continued speaking, slight cough, and scanty expectoration of grayish mucus tinged with dust or other impurities.

Laryngoscopic examination reveals redness and swelling of the vocal cords or of the entire larynx.

Treatment.—The use of the voice should be restricted. Coexisting nasal or pharyngeal disease should receive attention. The patient must learn to use the voice properly, expelling sounds by the abdominal muscles and diaphragm, and not by the muscles of the throat. Flannel protectors should be avoided, and the application of cool water to the neck, night and morning, instituted in their stead. Tonics are frequently indicated.

Thorough cleansing of the nose, throat, and larynx should be secured by means of mild alkaline sprays (Dobell's solu-

tion). Astringent sprays, such as the following are useful: Zinc acetate, 3 to 5 grains to the ounce; zinc sulphocarbolate, 2 to 3 grains to the ounce; alum, 3 to 5 grains to the ounce. Direct applications of silver nitrate (3 to 5 grains to the ounce) are also very efficacious.

Tuberculosis Laryngitis.—This may be primary, but it is usually secondary to tuberculosis elsewhere, especially in the lung.

Symptoms.—These consist in hoarseness, aphonia, hacking cough, and pain in the throat, increased by coughing, speaking, and swallowing.

Laryngoscopic Examination.—The mucous membrane is swollen, *pale*, and edematous. The arytenoid cartilages are especially involved, and the membrane between them is often the seat of a hill-like infiltration. Tuberculous ulcers are usually shallow and have a broad base, an irregular outline, and an uneven surface. They are extremely painful.

Treatment.—The general treatment should be that of pulmonary tuberculosis. The parts should be frequently cleansed with alkaline detergent sprays. Terebene, compound tincture of benzoin, or eucalyptol may be used in a respirator or inhaled from the surface of boiling water. Radical treatment consists in rubbing in, under cocain anesthesia, lactic acid (30 to 75 per cent. solutions). Palliative treatment consists in applying cocain in solution (4 to 10 per cent.) or orthoform, anesthesin, or iodoform in powder. The following insufflation is useful:

R. Orthoformi
Resorcinolis
Iodoformi..... āā ʒi.—M.

Syphilitic laryngitis may manifest itself as a catarrhal inflammation, mucous patches, gummatous infiltration, or ulceration. The ulcers are more or less circular, deep, and sharply circumscribed. They are frequently found on the epiglottis. Rapid necrosis and exfoliation of the cartilage may follow. Pain is often slight.

Diagnosis.—The history, the presence of other syphilitic lesions, the deep, clean-cut, rapidly spreading ulcers, the effect of treatment, and the absence of marked pain and of pulmonary lesions well serve to distinguish syphilis from *tuberculosis*.

Treatment.—Constitutional treatment with arsphenamin, iodids and mercurials is of the first importance. Local cleanliness should be secured by thorough spraying with some alkaline antiseptic solution. Ulcers may be touched with silver nitrate (melted on a silver probe), acid nitrate of mercury (1 to 5 parts of water), or chromic acid (1 to 8 parts of water). Insufflations of iodoform are also useful. Cicatricial stenosis may call for gradual dilatation or even tracheotomy.

LARYNGISMUS STRIDULUS

(Spasm of the Glottis; Laryngospasmus; "Child-crowing")

Definition.—A paroxysmal neurosis, characterized by reflex spasm of the adductors of the larynx, and not excited by any local inflammation.

Etiology.—It usually occurs in rachitic infants between six months and two years of age, and is apparently related to tetany. The attacks may be brought on by fright, gastric irritation, exposure, or a sudden movement.

Symptoms.—The attacks often occur on waking from sleep, and are characterized by a sudden arrest of breathing and tonic muscular spasms. The face is pale, and later cyanosed; the eyes are rolled up; the body is arched; the thumbs are turned into the palms; the legs are extended, and the soles turned inward. In a few seconds the spasm relaxes, and air is drawn through the glottis with a shrill, crowing sound.

The seizures vary greatly in frequency: several may occur in a day, or they may be weeks apart.

Diagnosis.—The intermittent character of the affection; the peculiar crowing inspiration; the absence of fever, cough, and hoarseness will serve to distinguish laryngismus from *spasmodic croup*.

Prognosis.—The prognosis is, as a rule, good. In the very young death may result from suffocation.

Treatment.—*The Paroxysm.*—Cold water may be dashed on the face and head, or a few drops of nitrite of amyl may be placed on a handkerchief and held before the nose.

The Interval.—Careful search should be made for some exciting cause; the gums may require lancing, or the gastrointestinal tract may demand attention. The child should be placed under the best hygienic conditions. The food should be plain and nutritious; tonics, as cod-liver oil, hypophosphites, and arsenic, are generally indicated. The bromid of sodium is an efficient antispasmodic, and may be advantageously combined with antipyrin:

R̄.	Sodii bromidi.....	ʒiss
	Antipyrinæ.....	gr. xx-xxx
	Glycerini.....	fʒss
	Aquæ menthæ piperitæ.....	fʒiij.

Sig.—A teaspoonful four times a day.

EDEMA OF THE LARYNX

(Edema of the Glottis)

Definition.—An infiltration of serous fluid into the sub-mucous tissue of the larynx.

Etiology.—It occasionally results from severe attacks of catarrhal laryngitis. It may be induced by severe inflammation of adjacent organs—as the tonsils, parotid glands, and pharynx. It may be a complication of some acute infectious disease, such as diphtheria, scarlet fever, or facial erysipelas. It is sometimes associated with ulcerative affections of the larynx, as tuberculosis and syphilis. It may be excited by the irritation of burns, scalds, or caustics. It occasionally occurs abruptly in the course of renal disease.

Pathology.—The connective tissue of the larynx is infiltrated with a serous or seropurulent fluid. The mucous membrane is tense and changed in color.

Symptoms.—These are: hoarseness of the voice, and later aphonia; extreme dyspnea, at first on inspiration, but later on expiration also; stridulous respiration; barking cough; and the evidences of dyspnea—namely, anxious face, blue lips, prominent sternocleidomastoids, and retraction of the base of the chest. When the epiglottis is involved, the swelling can be detected by the finger in the throat.

Laryngoscopic Examination.—The mucous membrane is swollen and of a reddish-purple color. The epiglottis may resemble a round, translucent tumor. In infraglottic edema the upper part of the larynx may appear normal, but swollen and edematous membrane is seen projecting through the glottis. The vocal cords are rarely affected.

Prognosis.—The condition is often fatal.

Treatment.—Mild inflammatory edema sometimes yields to the sucking of ice, local bloodletting, the application of ice to the neck, astringent sprays (alum, adrenalin, tannic acid), and the administration of saline purges. When the symptoms become urgent, the parts should be scarified under cocain anesthesia, and if this fails, tracheotomy should be performed at once.

DISEASES OF THE BRONCHI AND LUNGS

BRONCHITIS

Definition.—An inflammation of the bronchial tubes, characterized by substernal soreness, cough, mucopurulent expectoration, and dry and moist râles.

Varieties.—(1) Acute catarrhal bronchitis; (2) chronic catarrhal bronchitis; (3) fibrinous bronchitis.

ACUTE CATARRHAL BRONCHITIS

Etiology.—A cold, damp climate; changeable weather; occupations that necessitate confinement or the inhalation of irritating dusts or vapors; the gouty diathesis; and chronic heart disease are general predisposing factors.

In many cases the disease follows exposure to cold and wet, particularly when the body is overheated, or the inhalation of irritating gases or dusts. Not rarely it is one of the manifestations of a general infection such as measles, whooping-cough, typhoid fever, influenza, etc.

The exciting cause may be the *Micrococcus catarrhalis*, *pneumococcus*, *influenza bacillus*, *streptococcus* or *staphylococcus*.

Pathology.—In most cases the trachea and large tubes only are affected. The mucous membrane is red, swollen, injected, and covered in places with tenacious mucus.

Microscopic examination reveals desquamation of epithelium and infiltration of the submucous tissues with leukocytes.

Symptoms.—The chief features are: Chilliness and general malaise; a sense of soreness and constriction behind the sternum, increased by coughing; slight fever (100° to 102° F.), with its associated symptoms; and cough, which is at first dry and

painful, but later accompanied by more or less abundant mucopurulent expectoration.

Physical Signs.—If the inflammation is confined to the trachea and main bronchi there may be no abnormal signs. In other cases auscultation may reveal sonorous and sibilant râles on both sides of the chest in the early stages, and moist râles later when the bronchial secretion is more abundant.

Diagnosis.—*Influenza.*—High fever, severe pain in the head, back, and limbs, and great prostration will serve to distinguish influenza from bronchitis when the former is prevalent.

Catarrhal Pneumonia.—Moderately high and irregular fever, prostration, pronounced dyspnea, cyanosis, and physical signs indicating consolidation will aid in the recognition of pneumonia.

Prognosis.—The prognosis in general is good. In the aged the very young and the debilitated, however, the disease may lead to capillary bronchitis (catarrhal pneumonia).

Treatment.—If the patient be weak or old, he should be confined to his room or even to bed. At the outset, free diaphoresis is useful. It may be secured by means of hot drinks, a full dose of Dover's powder, and a hot foot-bath. Counterirritation to the chest in the form of sinapisms or stupes is very beneficial. The food should be simple and readily digestible, and the bowels should be kept regularly open by the aid of mild aperients. In the early stages, when there is no secretion, sedative expectorants—ipecacuanha, potassium citrate, and tartar emetic—are indicated. It is usually necessary to add a sedative, as opium or one of its derivatives (codein, $\frac{1}{8}$ to $\frac{1}{6}$ grain, or heroin, $\frac{1}{16}$ to $\frac{1}{10}$ grain), to allay the distressing cough. A combination such as the following will be found useful:

R.	Potassii citratis.....	ʒiij
	Vini ipecacuanhæ.....	fʒiiss
	Tincturæ opii camphoratæ..	fʒiij
	Succi limonis.....	fʒj
	Syrupi.....	q. s. fʒvj.—M.

SIG.—A tablespoonful every four hours.

When the secretion becomes more abundant, stimulant expectorants are indicated. One of the most reliable members of this class is ammonium chlorid; it may be prescribed in some simple vehicle, as brown mixture, or with squill, as in the following formula:

℞. Ammonii chloridi..... ʒiiss
 Syrupi scillæ..... fʒv
 Tincturæ opii deodorati..... ℥xl
 Extracti glycyrrhizæ..... ʒj
 Glycerini..... fʒss
 Aquæ..... q. s. ad fʒiv.—M.
 SIG.—A dessertspoonful in water every four hours.

Among other useful stimulant expectorants may be mentioned terpin hydrate, terebene, oil of eucalyptus, oil of santal, and tar. Such combinations as the following are often serviceable when the catarrh tends to become subacute:

℞. Ammonii chloridi..... ʒiss
 Terebeni..... fʒiss
 Codeinæ sulphatis..... gr. iii.—M.
 Pone in capsulas No. xxiv.
 SIG.—One every three or four hours.

Or:

℞. Ammonii chloridi..... ʒj
 Vini picis liquidæ
 Misturæ glycyrrhizæ compositæ..... āā fʒiss.—M.
 SIG.—A dessertspoonful every two or three hours. (MUSSEY.)

In the aged and infirm alcoholic stimulants may be required to combat general adynamia. Strychnin is a most valuable adjunct to the expectorants when there are indications that the heart is becoming strained by the violent paroxysms of cough. Should there be evidence of pronounced cardiac failure, it will be necessary to employ digitalis.

Such tonics as cod-liver oil, iodid of iron, quinin, and arsenic are often useful during convalescence from severe and prolonged attacks. Much benefit will also be obtained from suitable change of climate.

CHRONIC BRONCHITIS

(Chronic Bronchial Catarrh; Winter Cough)

Etiology.—This condition may be the result of repeated attacks of acute bronchitis, or it may develop gradually in association with chronic cardiac, pulmonary, or renal disease, or gout. It is especially common in the aged.

Pathology.—The mucous membrane of the bronchi is sometimes thickened and granular; in other cases it is thin from atrophic changes. The surface is usually covered with mucus; ulcers are occasionally noted.

Long-standing bronchitis leads to dilatation of the tubes (bronchiectasis) and to emphysema.

Symptoms.—The chief features are: Persistent cough with more or less mucopurulent expectoration; a sense of soreness behind the sternum. Fever is usually absent, and unless the disease is very severe, the general health may be fairly well preserved. Dyspnea on exertion is sometimes a troublesome symptom; it, however, belongs more to the resulting emphysema than to the bronchitis.

PHYSICAL SIGNS.—Unless emphysema has developed, inspection, palpation, and percussion give negative results.

Auscultation sometimes reveals râles, some of which are dry and wheezing, while others are moist and bubbling.

Bronchorrhea.—This term is applied to cases of chronic bronchitis which are associated with a very copious expectoration. The sputum is generally mucopurulent, and sometimes very offensive (fetid bronchitis).

Dry Catarrh.—This form, described by Laennec as *catarrhe sec*, is characterized by severe paroxysms of coughing with little or no expectoration. It is generally seen in the aged in association with emphysema or asthma.

Diagnosis.—*Pulmonary Tuberculosis.*—The absence of fever, of hemorrhage, of bacilli in the sputa, and of signs indicating consolidation will serve to distinguish chronic bronchitis from pulmonary tuberculosis.

Bronchiectasis.—This often results from chronic bronchitis. It is characterized by paroxysms of cough attended with the expectoration of large quantities of purulent secretion of an extremely offensive odor. There may be, also, physical signs of one or more cavities near the root or base of the lung.

Emphysema.—Marked dyspnea, distention of the chest, hyperresonance on percussion, and a prolonged feeble expiration on auscultation will indicate emphysema.

Sequelæ.—These comprise emphysema, bronchiectasis and dilatation of the heart, especially of the right ventricle.

Prognosis.—Complete recovery is, as a rule, not attainable, but the disease is not incompatible with long life.

Treatment.—Treatment must be directed toward the prevention of recurring acute attacks, and the removal, if possible, of the underlying cause. Change of climate, especially in winter, is most beneficial. If there is much secretion, a dry, warm climate is generally to be recommended, whereas if there be little expectoration, a moist warm climate is preferable. If patients cannot afford to travel, they should remain indoors as much as possible in bad weather, and take every precaution against chilling. Flannel should at all times be worn next to the skin, the feet should be kept dry, and night air should be avoided.

Underlying chronic diseases should receive appropriate treatment. If cardiac insufficiency is present, digitalis and strychnin will be required. If there is general malnutrition, such remedies as iron, arsenic, cod-liver oil, and hypophosphites may be given with advantage. If gout is a factor, iodids and alkalis will prove serviceable.

The most useful *direct remedies* are the stimulant expectorants, such as terebene, oil of eucalyptus, myrtol, oil of santal, oil of copaiba, oil of cubeb, and tar. If the sputum is heavy and purulent, no drug acts so well as creosote or the carbonate of guaiacol. Potassium iodid may be tried if the expectoration is scanty and viscid. Mild anodynes—heroin or codein—are often necessary to control harassing cough. The following

formulas will illustrate the manner in which these remedies may be combined:

℞. Terebeni
 Olei eucalypti
 Olei santali..... āā f3j-iss
 Codeinæ sulphatis..... gr. ii-j.—M.

Pone in capsulas No. xxiv.

SIG.—One after each meal and at bedtime.

℞. Terpini hydratis..... 3j
 Guaiacolis carbonatis..... 3ij
 Strychninæ sulphatis..... gr. ss
 Codeinæ sulphatis..... gr. ii-j.—M.

Pone in capsulas No. xxiv.

SIG.—One or two capsules three or four times a day.

℞. Apomorphinæ hydrochloridi..... gr. ss
 Syrupi pruni virginianæ..... f3ij
 Syrupi picis liquidæ..... f3iv.—M.

SIG.—A tablespoonful thrice daily. (MURRELL.)

Inhalations(eucalyptol, terebene, oil of Scotch fir, compound tincture of benzoin, etc.) are often efficacious. Such a mixture as the following may be employed several times a day in an oronasal respirator:

℞. Chloroformi..... f3ss
 Creosoti
 Terebeni
 Olei pini sylvestris..... āā f3iss
 Alcoholis..... q. s. ad f3j.

SIG.—From 5 to 20 drops to be used in the inhaler several times a day.

Counterirritation, preferably with iodine or small blisters, is often of great service in lessening the severity of acute exacerbations.

FIBRINOUS BRONCHITIS

Definition.—A rare affection characterized by the expectoration of fibrinous casts of certain portions of the bronchial tree.

Etiology.—The causes are unknown. In some cases it has been associated with tuberculosis, while in others there has been chronic heart disease.

Pathology.—The disease is often limited to a certain number of bronchi. Some of the affected tubes are found filled with a fibrinous exudate, while others are found empty and show a loss of epithelium. The casts are usually expelled in the form of whitish balls, which, when unrolled in water, present branching molds of the divisions and subdivisions of the affected bronchi. On close examination they are found to be hollow and laminated. Under the microscope, a homogeneous or fibrillated membrane is observed, imbedded in which are leukocytes, fat-drops, particles of pigment, epithelial cells, and occasionally Leyden's octahedral crystals.

Symptoms.—Acute and chronic forms are recognized. The former is rare, and manifests the symptoms of a severe attack of acute bronchitis, but the sputa contain fibrinous casts and there is marked dyspnea.

The chronic form is characterized by severe cough, paroxysms of dyspnea, and the expectoration of fibrinous plugs. Hemoptysis is not uncommon. The physical signs are those of chronic bronchitis and emphysema. The disease often lasts a few weeks, and then disappears, to return again at definite periods.

Prognosis.—In the acute variety the prognosis must be guarded; death frequently results from suffocation. In the chronic form attacks occur at intervals through many years.

Treatment.—In the acute cases the atmosphere of the room should be kept moist and uniformly warm. Inhalations of alkaline vapors (lime-water) appear to be beneficial. Counterirritants should be applied to the chest. Emetics sometimes aid in the expulsion of loose casts. Sedative or stimulant expectorants may be prescribed, as in catarrhal bronchitis. In the chronic form potassium iodid may also be given.

BRONCHIECTASIS

(Dilatation of the Bronchi)

Etiology.—Bronchiectasis is most frequently the result of chronic bronchitis, weakening of the walls of the bronchi from the inflammation and increased pressure from the violent coughing being the determining factors. In children it is sometimes secondary to bronchopneumonia. In other cases it develops as a sequel to chronic interstitial pneumonia, tuberculosis, or chronic dry pleurisy. Occasionally the disease is caused by obstruction to a bronchus (foreign body,

aneurysm, tumor, etc.), and in some cases it appears as a congenital defect.

Pathology.—Two forms are noted: (1) The cylindric form, in which the tubes, particularly those of medium size, are uniformly dilated in one or both lungs; and (2) the saccular form, in which the tubes swell out, here and there, into circumscribed dilatations that may reach several inches in diameter. Bronchiectatic cavities are lined with mucous membrane, but the latter is often atrophied, indurated, or ulcerated.

Symptoms.—The chief symptoms are paroxysmal cough, dyspnea, and copious expectoration. The last is characteristic. From time to time, especially in the morning or on a sudden change of posture, the patient expels a large amount (200–600 c.c.) of extremely fetid mucopurulent sputum, which on standing separates into three layers—an upper layer of discolored froth, a middle layer of turbid mucus, and an under layer of decomposed pus. Microscopically, it contains pus-corpuscles, fat crystals, crystals of hematoïdin, and numerous microorganisms, but not tubercle bacilli. Elastic fibers are rarely found. Hemoptysis is not uncommon.

PHYSICAL SIGNS.—In the cylindric variety the signs are those of chronic bronchitis. The saccular variety may present the signs of tuberculous cavities—localized tympany, cavernous breathing, gurgling râles, and pectoriloquy. Bronchiectatic cavities are usually near the root or the base of the lung. In long-standing cases the fingers are often clubbed.

Diagnosis.—*Pulmonary Tuberculosis.*—The marked constitutional symptoms, the apical location of the cavities, the signs of consolidation around the cavities, and the presence of tubercle bacilli and elastic fibers in the sputum will establish the diagnosis.

Prognosis.—There is little prospect of cure, but life may be prolonged indefinitely. Abscess or gangrene of the lung, metastatic abscesses, especially in the brain, and amyloid disease of the viscera are not uncommon sequels.

Treatment.—The general treatment is that of chronic bronchitis. The most useful expectorants are oil of eucalyptus, oil of santal, terebene, tar, guaiacol carbonate, and creosote. Inhalations of terebene, carbolic acid, creosote, etc., lessen cough and aid in destroying the fetid odor of the breath. Intratracheal injections ($\frac{1}{2}$ –1 dram of olive oil containing 1 or 2 per cent. of guaiacol) are sometimes efficacious. If a single large cavity can be definitely located in the lower lobe, incision and drainage may be considered.

BRONCHIAL ASTHMA

Definition.—Bronchial asthma is a condition characterized by paroxysmal attacks of dyspnea, in which the expiration especially is prolonged, the chest is over-distended, and usually there is expectoration containing characteristic spiril-like masses and many eosinophilic cells.

Etiology and Pathogenesis.—The disposition to asthma is sometimes hereditary. No age is exempt, but the large majority of cases begin in childhood or early adult life. Males are affected more frequently than females. Not rarely, the attacks are apparently related to infection of the upper respiratory tract or to nasal abnormalities. The essential etiologic factor is usually, if not invariably, abnormal sensitiveness to some specific foreign protein, the paroxysm itself being an anaphylactic or allergic reaction. The protein may be one contained in plant pollen (hay-fever), animal emanations (horse dandruff, cat-hair, feathers, etc.), food or bacteria. The chief pathologic conditions responsible for the attacks are spasm of the muscular coat of the bronchioles and congestive swelling of the bronchiolar mucous membrane.

Symptoms.—The paroxysms often appear suddenly, but in some cases certain symptoms precede and give warning of the approaching attack; among these are chilliness, flatulence, sneezing, and a copious discharge of pale urine. The attacks most often occur at night. There is a sense of op-

pression and anxiety, followed by dyspnea so intense that the patient runs to the window for air, or sits upright with his arms in such a position that he can bring into play the auxiliary muscles of respiration. The face is pale and anxious, the lips are blue, and the surface is covered with profuse perspiration. The respirations are not rapid, but labored and noisy. Cough is usually present, and is associated with the expectoration of thick, tenacious mucus. On close examination little grayish plugs can be detected in the sputum. These, under a pocket-lens, are seen to consist of delicate spirals of mucus that have been molded in the finer bronchioles (Curschmann's spirals). In addition, the sputum contains many eosinophilic leukocytes and Charcot-Leyden octahedral crystals. Examination of the blood usually reveals eosinophilia.

The paroxysms last, as a rule, two or three hours, and may recur for several successive nights, or may disappear entirely for weeks or months.

Physical signs are definite. During the attack the chest is fixed in full inspiratory expansion, and the abdomen is immobile. *Percussion* gives hyperresonance in long-standing emphysematous cases. On *auscultation* there are numerous sonorous and sibilant râles, especially during expiration, which is greatly prolonged and wheezy. Toward the end of an attack the râles become moist.

Diagnosis.—So-called *cardiac asthma* (*recurring pulmonary edema*) may be distinguished by the evidence of organic heart disease, absence of expiratory prolongation and absence of Curschmann's spirals and eosinophiles from the sputum.

Hay asthma may be recognized by the periodicity of the attacks and the associated coryza and sneezing.

Laryngeal Obstruction from foreign bodies, croup, paralysis of the vocal cords, or edema.—The dyspnea is with inspiration, and the chest, instead of being distended, is retracted, especially at the base.

In many cases the *type of sensitization* can be definitely determined by skin tests.

Sequelæ.—Emphysema invariably follows if the asthma is of long duration; it results from the tension to which the vesicles are subjected during the expiratory effort. Dilatation of the right ventricle is also a remote sequel.

Prognosis.—The disease does not prove fatal except through complications or sequelæ. Complete recovery may occur in young subjects if the exciting cause can be found and removed.

Treatment.—*The Interval.*—The cause must be sought for in every case, and removed if possible. Desensitization by subcutaneous injections of the offending protein (except food proteins) is often of great benefit. In the case of food sensitization relief is afforded by keeping the offending protein out of the diet. Digestive disturbances should always receive careful attention. Chronic bronchitis, emphysema, and dilatation of the heart are frequent concomitants of asthma and call for special treatment. Change of climate, even though slight, usually proves of decided service, but the choice of locality must be determined very largely by the personal experience of the patient. Many sufferers do better in the smoky atmosphere of cities than in the country, but a dry atmosphere with a moderate elevation is better suited to the majority.

Among internal remedies none has proved more useful than potassium iodid (5 to 10 grains thrice daily) in averting attacks. Tincture of belladonna (3 to 5 minims thrice daily) is a valuable adjunct to the iodid. Arsenic may be tried if the iodids fail. Grindelia is sometimes useful if there is much catarrh. Strychnin is of service in cases associated with emphysema.

The Attack.—Some patients derive great benefit from the fumes of ignited stramonium or belladonna leaves or paper that has been impregnated with potassium nitrate. These agents may be burnt in the patient's room or smoked in a pipe or in the form of cigarettes. Marked alleviation of the paroxysms is often obtained from the inhalation of amyl

nitrite (5 to 6 minims), ethyl iodid (10 to 20 minims), or a few whiffs of chloroform. If such measures fail to afford relief, internal remedies must be used. In some cases strong hot coffee acts most happily; in others more benefit is derived from hot whisky and water. Among the numerous special remedies that have been advocated the following appear to be the most reliable: opium, belladonna, bromids, chloral, paraldehyd, Hoffmann's anodyne, lobelia, and quebracho.

Few attacks will resist the action of morphin with atropin hypodermically, but the greatest caution must be exercised in order that the patient may not become addicted to the drug. Heroin hydrochlorid hypodermically, in doses of from $\frac{1}{12}$ to $\frac{1}{10}$ grain, may often be substituted for morphin with advantage but, of course, it is also a habit-producing drug. In many cases hypodermic injections of adrenalin (5-15 minims) are very serviceable. When the attacks are associated with bronchial catarrh, a combination such as the following sometimes proves efficacious:

R̄.	Tincturæ belladonnæ.....	f 5j
	Tincturæ lobeliæ.....	f 5iij
	Fluidextracti aspidospermatis.....	f 3ss
	Strontii bromidi.....	3iiss
	Elixiris aromatici.....	q. s. ad f 3iv.—M.
SIG.—A dessertspoonful in water every two or three hours.		

Among other measures that have been found useful in alleviating asthmatic attacks may be mentioned the application of sinapisms to the chest, the inhalation of compressed air, and the inhalation of oxygen.

HAY ASTHMA

(Hay Fever; Autumnal Catarrh; Rose-cold)

Definition.—A catarrhal affection of the upper air-passages, characterized by coryza and asthmatic seizures, and evoked by inhaling the pollen of certain plants and grasses.

Etiology and Pathogenesis.—An inherited tendency, a nervous temperament, and chronic nasal disease are pre-

disposing factors. The attacks, as a rule, occur in the autumn (autumnal catarrh) or in the spring (rose-cold), and are excited by certain plants and grasses (ragweed, goldenrod, timothy grass, etc.).

The disease appears to be a local manifestation of hypersensitiveness (anaphylaxis) to the toxalbumin of the pollen (see p. 272).

Symptoms.—Nasal obstruction with rhinorrhea and much sneezing, congestion of the conjunctivæ with lacrimation, itching of the eyelids, nose, and palate, headache and lassitude, and, not rarely, paroxysms of asthma, constitute the symptoms.

“Rose-cold” usually begins in May or June and lasts to the latter part of July; “autumnal catarrh” begins in the latter part of August and ends with the first frost.

Prognosis.—The disease never proves fatal, but complete cure is not often observed. As age advances, however, the attacks not rarely become less severe.

Treatment.—Careful search should be made for chronic nasal disease, and, if found, appropriate treatment instituted.

A change of climate during the period of susceptibility exempts most patients. A sea-voyage or a sojourn in some high-mountain district, as the White Mountains, Adirondacks, Catskills, or Alleghenies, may be recommended. Frequently, desensitization of the patient by subcutaneous injections of the offending pollen, beginning the treatment at least three months ahead of the season of pollination, yields excellent results.

Tonics, such as quinin, arsenic, and strychnin, are often very useful when administered before and during an attack. To allay itching and lacrimation the eyes may be washed with a solution of boric acid (10 grains to the ounce) or sulphate of zinc (1 to 2 grains to the ounce). Sneezing, nasal fulness, and discharge are often relieved by medicated sprays (Dobell's solution) or the application, on pledgets of cotton, of adrenalin solution (1:5000).

PULMONARY EMPHYSEMA

Definition.—Abnormal distention of the lungs with air.

Varieties.—(1) *Interlobular Emphysema*.—This form is rare, and results from the rupture of the air-vesicles and the escape of air into the interstitial tissue.

(2) *Compensatory Emphysema*.—This is a vicarious distention of one part of the lung, owing to pathologic changes in another part of the organ. It is primarily physiologic, though atrophy of the walls of the air-vesicles may ultimately ensue.

(3) *Atrophic or Senile Emphysema*.—In this form the capacity of the air-vesicles is relatively increased, owing to atrophy of the solid tissue.

(4) *Hypertrophic or Substantive Emphysema*.—This is the ordinary form of emphysema. It is characterized by a great enlargement of the lungs in consequence of overdistention of the air-vesicles.

Compensatory emphysema, atrophic emphysema, and hypertrophic emphysema together form a subdivision known as vesicular emphysema.

HYPERTROPHIC EMPHYSEMA

Definition.—A pulmonary disease characterized anatomically by dilatation of the air-vesicles and atrophy of their walls; and clinically by permanent enlargement of the thorax, with more or less dyspnea.

Etiology.—Congenital weakness of the lung structure—probably a defective development of elastic tissue—may be important predisposing factor. This predisposition may be transmitted through several generations.

In forced expiration the air cannot escape with sufficient rapidity through the narrow glottis, and the backward pressure stretches the air-vesicles; hence the obstinate cough of chronic bronchitis, the expiratory straining of asthma, and

occupations that necessitate forced expiration, as playing on wind-instruments and glass-blowing, are causal factors.

Pathology.—The lungs are enlarged and do not collapse when the thorax is opened. In the advanced cases the free margins are studded with large bullæ or blebs that have resulted from the rupture of a number of vesicles into a common sac. The organs are pale and have a soft, cotton-like feel. Microscopic examination reveals atrophy of the vesicular walls, a diminished amount of elastic tissue, and more or less obliteration of the pulmonary capillaries. This last condition leads to increased tension in the pulmonary artery and ultimately to enlargement of the right ventricle.

Symptoms.—The disease usually manifests itself in middle life, but it is sometimes observed in the young. Dyspnea, at first only upon exertion, but as age advances, more or less persistent; a disposition to bronchitic attacks with cough and expectoration upon the slightest exposure; and cyanosis, often extreme during attacks of acute bronchitis, are the usual symptoms. In advanced cases edema of the feet may result from cardiac failure.

PHYSICAL SIGNS.—The neck is short, and the sternocleidomastoids are prominent. The thorax is likewise short but broad, especially in its anteroposterior diameter. This configuration has given rise to the term “barrel-shaped” chest. On respiration there is little expansion, but an elevation of the thorax as a whole. The apex-beat is usually invisible, but an abnormal pulsation is often noted in the epigastrium.

Palpation.—Vocal fremitus is usually diminished.

Percussion.—This yields hyperresonance all over the chest. The upper level of hepatic dulness is depressed, and the area of cardiac dulness may be almost obliterated.

Auscultation.—In uncomplicated emphysema the breath sounds are feeble and the expiration is much prolonged. In the presence of bronchitis, however, the breath sounds may be harsh or marked by coarse râles. The pulmonary second sound is often accentuated.

Complications.—The most important complications are: Bronchitis, asthma, dilatation of the right ventricle, and, later, tricuspid regurgitation and dropsy.

Diagnosis.—*Chronic Bronchitis.*—In this disease there is no marked dyspnea; the chest is not enlarged; there is no change in the percussion-note or in the expiratory sound.

Pneumothorax.—This disease develops suddenly, is unilateral, and yields a tympanitic note on percussion and, frequently, metallic tinkling and bell-tympany on auscultation.

Prognosis.—Emphysema is incurable, but its advance may be stayed by relieving the primary condition. It runs a long course and is in itself rarely fatal, but death may result from heart-failure and dropsy, or from intercurrent pneumonia.

Treatment.—The treatment advocated in chronic bronchitis and asthma is often applicable here. The patient should be placed under the most favorable hygienic conditions. Iron is indicated in the anemic. Strychnin ($\frac{1}{40}$ to $\frac{1}{30}$ grain) is a valuable respiratory and cardiac stimulant, and may be combined with digitalis when there are symptoms of heart-failure.

R. Strychninae sulphatis..... gr. ss
 Pulveris digitalis
 Pulveris scillae
 Ferri reducti..... āā gr. xx.—M.
 Fiant pilulae No. xx.
 Sig.—One thrice daily.

The inhalation of oxygen, or the inspiration of compressed air, followed by expiration into rarefied air, is sometimes a useful measure.

HEMOPTYSIS

(Bronchorrhagia; Bronchopulmonary Hemorrhage)

Definition.—The expectoration of blood.

Etiology.—The chief causes are: (1) Traumatism. (2) Certain organic diseases of the lung, especially tuberculosis

(50 per cent. of the cases), lobar pneumonia, bronchiectasis, gangrene, infarct, and cancer. (3) Passive congestion the result of heart disease, especially mitral lesions. (4) Aortic aneurysm, from erosion of the trachea or a bronchus, or rupture of the sac into the respiratory tract. (5) Diseases profoundly affecting the blood, such as purpura, hemophilia, scurvy, and leukemia. (6) Ulcers—traumatic, syphilitic, or malignant—of the trachea or larynx. (7) Vicarious menstruation (very rare).

Symptoms.—Sometimes the bleeding is preceded by cough, dyspnea, or substernal tenderness, but often there is no premonition, and the first indication is the presence of a warm salty fluid in the mouth. The blood is generally raised by coughing, and is bright red and frothy. It is alkaline in reaction, and intimately mixed with air and mucus. The hemorrhage is rarely profuse unless it results from the rupture of an aortic aneurysm or the ulceration of a large vessel in advanced pulmonary tuberculosis. Auscultation of the chest reveals bubbling râles. The subsequent expectorations are tinged with blood, and if much is swallowed, it may excite vomiting or pass into the intestine and impart a tarry appearance to the stools.

Diagnosis.—The differential diagnosis between *hematemesis* and *hemoptysis* has been considered on page 67.

Prognosis.—This depends upon the cause. It is rarely fatal, except in aneurysm and in advanced tuberculosis with a large cavity.

Treatment.—Absolute rest is essential. An ice-bag may be placed over the suspected seat of the hemorrhage, but it should be removed if it aggravates the coughing. Bits of ice may be given to the patient to suck. There is no more useful remedy than morphin, which serves to allay excitement and to check cough. It is best given hypodermically. Nitroglycerin (1 drop every half hour) has been strongly recommended. If the hemorrhage is protracted, a saline purge may be useful. Among other remedies that seem to be of service may be mentioned oil of erigeron, fluid extract of hamamelis,

and gelatin. Ergot is useless, and so is the inhalation of vaporized solutions of astringent drugs. The application of firm ligatures to the limbs may prove efficacious by lowering the intrapulmonary pressure.

HEMORRHAGIC INFARCT OF THE LUNG

(Pulmonary Apoplexy)

Definition.—A circumscribed area of necrosis in the lung, with extravasation of blood, caused by the occlusion of a branch of the pulmonary artery.

Etiology.—The most common cause of pulmonary infarct is obstruction of a branch of the pulmonary artery by an embolus coming from the right heart or the general venous system. In some cases the obstruction is caused by a thrombus, the formation of which has been favored by cardiac weakness.

Pathology.—The infarct is usually located in the periphery of the lung; it is conic in shape, with its apex pointing inward. The portion affected is firm, airless, and of a dark red color. Microscopic examination shows a dense aggregation of blood-corpuscles.

In small infarcts the dead tissue and blood are slowly absorbed and replaced by a cicatrix.

Symptoms.—When the infarct is large, the usual symptoms are localized pain, dyspnea, cough, and the expectoration of dark non-aërated blood. These symptoms occurring in chronic heart-disease or phlebitis are especially suggestive. Small infarcts occasion no *physical signs*; larger ones may yield a circumscribed area of dulness, with subcrepitant râles and feeble breath sounds, or, perhaps, bronchovesicular breathing.

Treatment.—The condition itself is not amenable to treatment. Remedies should be directed to the primary disease.

CONGESTION OF THE LUNGS

ACTIVE CONGESTION

Etiology.—This results from an increased afflux of blood to the lungs. Violent exercise, mountain climbing, and the

inhalation of irritants may produce it. It is an associated condition in all severe inflammatory diseases of the lungs. In the vast majority of cases it marks the initial stage of croupous pneumonia.

Pathology.—The lung is bright red in color, heavy, and less crepitant. When incised and pressed, copious frothy blood exudes.

Symptoms.—The chief symptoms are dyspnea; a short, dry cough, followed by frothy, blood-streaked sputum; and a rapid, full pulse. The occurrence of a chill and fever indicates commencing pneumonia.

Physical examination reveals slight dulness, crepitant râles, and bronchovesicular breathing.

Treatment.—The measures most likely to effect depletion of the lung are complete rest, the application of dry or wet cups to the chest, and the administration of veratrum viride and a saline purge.

PASSIVE CONGESTION

Etiology.—This results from obstruction to the flow of blood from the lungs to the heart. The chief cause is cardiac disease, especially lesions of the mitral valves and weakness of the left ventricle from fatty or fibroid changes.

Pathology.—The lungs are dark red in color, and often somewhat edematous. When the condition has lasted a long time, the organs become brown, dense, and tough (*brown induration*). Microscopic examination reveals dilatation of the capillaries, overgrowth of connective tissue, brown pigmentation, and degenerative changes in the blood-vessels.

Symptoms.—Dyspnea, cough, and the expectoration of blood-stained mucus containing pigmented epithelial cells (heart-disease cells) are the characteristic symptoms. *Physical examination* reveals slight dulness, feeble breathing, and abundant fine râles.

Treatment.—Remedies should be directed to the underlying cardiac disease. The application of dry or wet cups often

gives temporary relief. If the symptoms are urgent, venesection is indicated. Saline and mercurial aperients are of service.

HYPOSTATIC CONGESTION

Definition.—A congestion of dependent portions of the lungs occurring in asthenic diseases that necessitate a protracted recumbent position.

Etiology.—It is generally observed in prolonged febrile diseases, such as typhoid fever, and in adynamic states. Cardiac weakness, recumbent posture, and alterations in the blood are the causal factors.

Pathology.—The lungs are dark red and edematous posteriorly. The edema and increased amount of blood render the organs more solid and less crepitant. They never show the granular appearance of croupous pneumonia.

Symptoms.—The symptoms are often indefinite. There may be moderate dyspnea, slight cyanosis, cough, and, perhaps, blood-tinged expectoration.

Physical examination reveals dulness over the lower lobes, subcrepitant râles, and feeble, blowing breathing.

Treatment.—Efforts should be made to prevent the development of hypostatic pneumonia in asthenic diseases by frequent change of posture and the timely administration of such stimulants as strychnin, digitalis, alcohol, ammonia, and camphor. If already present, relief is sometimes afforded by dry or wet cupping or the application of stupes.

EDEMA OF THE LUNGS

Definition.—An effusion of serous fluid into the air-vesicles and interstitial tissue of the lungs.

Etiology.—Pulmonary edema may occur under the following conditions: (1) In passive congestion of the lungs, the result of chronic heart disease; (2) as a part of general dropsy induced by nephritis; (3) as a terminal process in many acute and chronic disorders; (4) in ether anesthesia; (5) after thoracentesis; (6) as an acute disorder in chronic disease of

the heart, arteries, or kidneys and in certain infectious diseases; (7) around circumscribed lesions of the lung, such as pneumonia, tuberculosis, infarct, etc. (collateral edema).

Pathology.—The lungs, especially the dependent portions, are heavy, red in color, and boggy to the feel. When the affected portion is incised and subjected to pressure, abundant blood-stained, frothy-serum exudes.

Symptoms.—These consist in dyspnea sometimes amounting to orthopnea, cyanosis, cough, and the expectoration of large quantities of frothy, serous fluid. Occasionally the sputum is blood-stained. The skin is often cold and livid. There is no fever.

Physical examination reveals feeble tactile fremitus, dullness, weak breath-sounds, and numerous fine and coarse, moist râles.

Diagnosis.—*Lobar Pneumonia.*—This is characterized by a chill, fever, pain, rusty expectoration, and signs of consolidation.

Hydrothorax.—In this condition there may be enlargement of the affected side, with displacement of the apex-beat. The upper level of dullness is often movable, and frothy sputum and râles are absent.

Prognosis.—This is always grave. It is often a terminal symptom of the disease in which it occurs. If not far advanced, and the primary disease is amenable to treatment, recovery may follow.

Treatment.—If there is much cyanosis and the patient's strength will permit it, the application of wet cups to the chest or bleeding from the arm may be of value. Sinapisms should be applied to the chest. Hydragogue cathartics (Epsom salts) are often indicated. Circulatory stimulants, such as digitalis, strychnin, caffeine, and camphor, are generally indicated and may be given hypodermically. Atropin ($\frac{1}{100}$ grain), hypodermically, is sometimes of service. Morphin ($\frac{1}{6}$ to $\frac{1}{4}$ grain subcutaneously) is invaluable in acute paroxysmal edema occurring in chronic cardiovascular disease, but it

is contraindicated in other forms. Inhalations of oxygen may be employed to lessen the dyspnea.

BRONCHOPNEUMONIA

(Capillary Bronchitis; Catarrhal Pneumonia; Lobular Pneumonia)

Definition.—An inflammation of the terminal bronchioles and air-vesicles.

Etiology.—It is most frequently observed in young children and in the aged. It is a common sequel of the specific fevers, especially of whooping-cough, measles, influenza, and diphtheria. In infants and in debilitated subjects of any age it may occur as a primary affection, the result of chilling.

Another group of cases results from the aspiration of infectious materials or particles of food into the smaller bronchi (aspiration or deglutition pneumonia). This accident is liable to occur whenever the sensibility of the larynx is benumbed, as in apoplexy, bulbar palsy, or uremia. Cancer of the throat and operations on the upper air-passages also favor its occurrence.

The immediate cause is some bacterium. The organism most frequently found is the *Diplococcus pneumoniae*. This may occur alone or in combination with the streptococcus, staphylococcus, bacillus of Friedländer, or more rarely with the typhoid bacillus, influenza bacillus, colon bacillus, or diphtheria bacillus.

Pathology.—As a rule, both lungs are involved. On section, small projecting areas of consolidation are noted here and there around the finer bronchioles. Recent patches are reddish-brown in color, firm, and smooth or finely granular; later they become grayish and soft. The terminal bronchi are filled with purulent material.

In addition to these solidified areas there are other small patches of collapsed lung that are airless, firm, and bluish-red in color. The collapse has resulted from occlusion of the bronchus, and closely resembles consolidation; but it

can, as a rule, be overcome when inflation is practiced by means of a blowpipe inserted in the supplying bronchus.

Microscopic examination reveals an exudate in the terminal bronchi and air-cells, which is composed of leukocytes and desquamated epithelium in various stages of degeneration. The walls of the bronchioles are also infiltrated with leukocytes.

Compared with croupous pneumonia, the contrast is marked. In the latter the lung is involved *en masse*; the consolidation is distinctly granular, and the exudate is composed of red blood-corpuscles, white blood-corpuscles, and fibrin; the lining epithelium is but slightly involved, and the walls of the bronchi are not infiltrated with leukocytes.

Terminations and Sequels.—(1) Resolution; the exudate undergoes autolytic digestion, and is eventually absorbed or expectorated. (2) Death frequently occurs from toxemia and exhaustion. (3) Termination in tuberculosis was supposed formerly to occur very frequently; it is now regarded as being relatively rare. Most of the cases in which this termination is supposed to have occurred were in reality cases of primary tuberculous pneumonia. (4) Pleurisy (fibrinous or purulent) sometimes follows. (5) Abscess and gangrene are common terminations in the aspiration form, but are rare in other types of the disease. (6) Chronic interstitial pneumonia is an occasional sequel.

Symptoms.—The symptoms are often masked by the primary disease. The onset is usually gradual, and is characterized by prostration, cough, and fever. The last is moderately high and very irregular (102° – 104° F.). The dyspnea is marked, and the respirations are rapid, in children often 40 to 50 a minute; the pulse is also accelerated—110 to 150 a minute; cough is painful and accompanied by a mucopurulent expectoration that is occasionally blood-streaked. The face is usually pale and anxious, and the lips may be blue.

PHYSICAL SIGNS.—As the areas of consolidation are generally small and scattered, the physical signs are inconstant.

Inspection may reveal evidences of dyspnea—lividity, playing of the nostrils, prominence of the sternocleidomastoids, etc.

Palpation usually gives negative results.

Percussion may reveal areas of dullness in one or both lungs.

Auscultation may reveal patches of subcrepitant râles or, perhaps, areas over which the breathing is bronchial or bronchovesicular.

Diagnosis.—*Acute Pulmonary Tuberculosis.*—In this disease there is a tuberculous bronchopneumonia which is difficult to distinguish from simple bronchopneumonia. A family history of tuberculosis, extensive involvement of the apices, bubbling râles indicating softening, the long duration, profuse sweats, rapid emaciation, and the presence of tubercle bacilli and elastic fibers in the sputa are the diagnostic phenomena of tuberculosis.

The following table will show the clinical differences between *bronchopneumonia* and *croupous pneumonia*:

BRONCHOPNEUMONIA	CROUPOUS PNEUMONIA
Often secondary to bronchitis or an acute infectious disease.	Usually a primary disease.
The onset is gradual and without a distinct chill.	The onset is abrupt and with a distinct chill.
The fever is moderately high, very irregular, and ends by lysis after an indefinite period, sometimes of two or three weeks' duration.	The fever is high, regular, and generally ends by crisis between the sixth and ninth day.
The sputum is mucopurulent or glairy and tenacious.	The sputum is rusty and translucent.
Both lungs are commonly affected.	In the majority of cases only one lung is affected.
The physical signs are indistinct and indicate scattered areas of consolidation.	The physical signs are distinct and indicate a large uniform consolidation.

Bronchitis.—In simple bronchitis the fever is not high, the dyspnea is slight, there is little prostration, and there are no signs of consolidation.

Prognosis.—In previously healthy children the prognosis is good. In cachectic children the outlook is very grave. Aspiration pneumonia is usually fatal. The average mortality in childhood is probably not less than 30 per cent.

The duration of the disease is usually from two to three weeks; a longer duration should suggest tuberculosis.

Treatment.—Much can be done by careful management in preventing bronchitis from gaining access to the smaller bronchi.

On the supervention of bronchopneumonia the patient should be confined to bed, in a well-ventilated room, which should be kept preferably at a temperature of about 65° F. The diet should be liquid and nutritious. Alcohol is often required in severe cases. From 15 to 20 minims of whiskey every three hours for a child of three years is usually sufficient.

At the outset it is advantageous to administer a mild laxative, preferably calomel or castor oil. If there is a harsh, dry cough, the application of the tincture of iodine of suitable strength usually affords some relief. In adults sinapisms or stupes may be used instead of the iodine.

Fever is best controlled by cold. Compresses wrung out of cold water may be wrapped around the chest and changed for fresh ones at intervals of twenty minutes. Expectorants are usually required. In the early stage potassium citrate is very serviceable. It may often be combined advantageously with spirit of nitrous ether and ammonium acetate, as in the following formula:

℞. Potassii citratis..... ʒij
 Spiritus ætheris nitrosi..... fʒv
 Liquoris ammonii acetatis..... fʒj
 Syrupi tolutani
 Aquæ..... āā q. s. ad fʒiv.—M.

SIG.—A dessertspoonful in water every three hours for a child of 4 years.

Later, the ammonium salts, especially the carbonate, are more efficacious. From 1 to 2 grains of the latter may be

given every three or four hours to a child of two years. Ammonium iodid is also useful, and may be employed as an adjuvant, as in the following formula:

℞. Ammonii carbonatis..... gr. xlvijj
 Ammonii iodidi..... gr. xxiv
 Syrupi tolutani
 Aquæ..... āā q. s. ad fʒiij.—M.
 SIG.—Teaspoonful every two or three hours for a child of three years.

If the child is unable to expel the mucus and the breathing becomes much oppressed, an emetic (ipecac) may prove of service. Inhalations of oxygen sometimes make breathing easier. Strychnin is also of benefit at this time in combating respiratory failure.

If symptoms of circulatory failure appear, digitalis must be given in addition to alcohol and strychnin. Extreme restlessness and insomnia will sometimes require the use of the bromids or some other mild sedative.

Except at the onset, when they may be necessary to relieve pleuritic pain and to control harrassing cough, opiates should not be used.

During convalescence tonics and change of air are often required.

CHRONIC INTERSTITIAL PNEUMONIA

(Cirrhosis of the Lung; Chronic Pneumonia; Pulmonary Induration)

Definition.—A chronic disease of the lung, characterized by an overgrowth of fibrous tissue.

Etiology.—It is rarely a sequel of lobar pneumonia or of bronchopneumonia. It may be excited by the constant inhalation of irritating dusts, as stone-dust (chalicosis), coal-dust (anthracosis), or metal-dust (siderosis). It may result from syphilis. It is occasionally secondary to chronic pleurisy. It is an invariable accompaniment of chronic tuberculosis.

Pathology.—When the thorax is opened, the lung is found retracted and the heart displaced. The organ is tough,

firm, and more or less airless. Section shows an overgrowth of fibrous tissue, and usually inflammation and considerable dilatation of the bronchi.

Symptoms.—The chief symptoms are dyspnea on exertion and cough. The latter may be dry, but it is usually associated with more or less mucopurulent sputum. There is rarely fever, and the general health may be well preserved for many years.

PHYSICAL SIGNS.—*Inspection* may reveal retraction of the affected side and displacement of the apex-beat of the heart.

Percussion may yield dulness. Over saccular dilatations of the bronchi there may be a tympanitic note.

Auscultation.—The vocal resonance is increased, and the breathing is often bronchial or even cavernous.

Diagnosis.—*Fibroid Phthisis.*—This is less likely to involve the bases, fever is a frequent accompaniment, and tubercle bacilli are present in the sputa.

Prognosis.—The disease is incurable. Its course, however, is extremely chronic. Death may be due to intercurrent disease or dilatation of the right heart.

Treatment.—This is largely hygienic and coincides with that laid down for tuberculosis. Stimulant expectorants are useful when bronchitis or bronchiectasis is a prominent feature.

ABSCESS OF THE LUNG

Etiology.—(1) It is most frequently caused by the aspiration of infective material from the upper respiratory tract (ether narcosis, tonsillectomy, etc.) (2) It may result from the impaction of a foreign body in a large bronchus. (3) It may arise from the extension of a suppurative process in an adjacent structure, such as the pleura or liver. (4) It is occasionally of traumatic origin. (5) It is rarely a sequel of lobar or lobular pneumonia. (6) Multiple abscesses of embolic origin are common in malignant endocarditis and pyemia.

Symptoms.—Remittent or intermittent fever, rigors, sweats, pallor, and leukocytosis indicate suppuration. Dyspnea, cough, and purulent, offensive sputa containing shreds of lung tissue are the pulmonary symptoms. Physical examination may reveal bubbling râles, and, later, cavernous breathing and pectoriloquy. Multiple embolic abscesses are rarely recognized during life.

Prognosis.—Many cases following pneumonia and the rupture of external abscesses into the lung end in recovery. Embolic abscess always proves fatal.

Treatment.—Nutritious food and quinin, strychnin, and alcoholic stimulants will be required to support the system. Single abscesses, if they can be localized, should be opened and drained.

GANGRENE OF THE LUNG

Etiology.—Gangrene of the lung is not a primary condition, but is secondary to inflammation or necrosis of the lung tissue. It is excited by the entrance of bacteria of putrefaction, but unless the system is considerably reduced in vitality, the tissues, even though diseased, show remarkable resistance and escape putrefaction.

Pneumonia, especially aspiration-pneumonia, tuberculosis, pressure of morbid growths, bronchiectasis, abscess, and hemorrhagic infarction following embolism of the pulmonary artery are the predisposing pulmonary conditions. Nephritis, alcoholism, the infectious fevers, and particularly diabetes, by lowering vitality, render the lung more liable to be attacked.

Pathology.—The process may be circumscribed or diffuse. The affected part is converted into a greenish-black, soft mass having an extremely fetid odor. After the softened material has been expectorated, there is left behind a cavity with ragged walls, containing a foul-smelling liquid. The tissues around the cavity are inflamed and edematous.

Symptoms.—Persistent cough, irregular fever, and emaciation are usually present. Hemoptysis is common. The expectoration is characteristic; it is profuse, and has a pene-

trating, offensive odor. When allowed to stand in a glass vessel, it separates into three layers: a frothy layer on top, a translucent serous layer in the middle, through which hang strings of pus, and at the bottom a layer of reddish-green purulent material. Altered blood may give it the appearance of prune-juice. Microscopically it contains shreds of tissue, crystals of fatty acids, crystals of hematoidin, and numerous pyogenic bacteria.

The *physical signs* are those of solidification followed by those of excavation. Physical signs of pyopneumothorax may supervene from perforation into the pleura.

Prognosis.—This is grave, but not hopeless. Death may result from exhaustion, hemorrhage, or cerebral abscess the result of embolism.

Treatment.—Nutritious food and strychnin, quinin, and alcohol are required to support the system. Inhalations of creosote or of formalin (2 per cent. gradually increased to 5 per cent.) may be employed to lessen the fetor of the breath. Surgical intervention is indicated if the gangrenous process can be localized and is not a complication of an incurable disease.

TUMORS OF THE LUNGS AND PLEURA

Intrathoracic tumors are almost always malignant. Carcinoma may be primary, but it is usually secondary. Sarcoma, with rare exceptions, is secondary. Pleural endotheliomata have been somewhat frequently reported. The chief *symptoms* may be those of a progressive solidification of the lung, of stenosis of the larger bronchi or trachea or of pleurisy with effusion. Cough, dyspnea, expectoration of bloody material, hemoptysis, pleuritic pain and cachexia are present in most of the cases. Solidified areas yield dullness on percussion and feeble or bronchial breathing, according as the bronchi are pervious or obstructed. The x-ray is often a valuable aid in diagnosis. Pleural effusion of neoplastic origin is usually bloody and returns rapidly after thoracentesis. In doubtful cases the presence of malignant disease elsewhere in the body, the location of the dull areas or the character of the effusion, the cachexia, and the absence of tubercle bacilli from the sputum on repeated examination are the important diagnostic points. The *duration* intrathoracic malignant disease is from a few weeks to two or three years. The *treatment*, as a rule, can only be palliative.

DISEASES OF THE PLEURA

PLEURISY

(Pleuritis)

Definition.—Inflammation of the pleura.

Varieties.—According to cause it may be divided into primary or secondary; according to extent, into unilateral, bilateral, or localized; according to duration, into acute or chronic; and according to the exudation, into serofibrinous, fibrinous, or purulent.

Etiology.—Pleurisy may be: (1) Idiopathic, arising from exposure to cold and wet; (2) secondary to inflammatory diseases of adjacent structures, such as pneumonia, pulmonary tuberculosis, etc.; (3) secondary to general morbid processes that lessen tissue resistance, such as the specific fevers, chronic nephritis, cancer, etc.; (4) traumatic; (5) tuberculosis. *At least three-fourths of all cases of so-called idiopathic pleurisy are tuberculous, and in at least one-fourth of such cases definite signs of pulmonary involvement sooner or later develop.* The bacteria most commonly found in the exudate are the tubercle bacillus, pneumococcus, and streptococcus.

Pathology.—In the early stage the membrane is red, sticky, lusterless, and covered with a thin film of fibrin; if the process now ceases, the condition is termed *dry pleurisy*. If, however, the inflammation continues, an exudate is formed which may be: (1) Serofibrinous; (2) fibrinous; or (3) purulent (empyema). In the *serofibrinous* form there is little fibrin, the exudate being mainly composed of straw-colored serum (a few ounces to several pints), which in favorable cases is gradually absorbed. In large effusions the adjacent organs

are displaced and the lungs are compressed. In the *fibrinous* form serum is scanty and the membrane is covered with a butter-like exudate that subsequently becomes organized and unites more or less closely the pleural surfaces, causing *chronic pleural thickening*. If the effusion is liquid and confined to pockets formed by adhesions, the condition is termed *sacculated pleurisy*.

Purulent pleurisy commonly results from the extension of infection from a contiguous structure, especially the lung: (1) It may cause general sepsis; (2) it may rupture spontaneously into the lung, through the chest-wall (*empyema necessitatis*), or, very rarely, into the esophagus, pericardium, stomach, or peritoneum; (3) the pus at times may be slowly absorbed or become inspissated and calcified. Gradual recovery is not infrequent after perforation of the chest-wall, and is occasionally observed after perforation of the lung. In favorable cases, after the discharge of the pus, the pleural surfaces eventually become united by firm adhesions.

Hemorrhagic Pleurisy.—A bloody effusion is often observed in tuberculous and cancerous pleurisies and in pleurisy associated with scurvy, grave anemia, and other cachectic states.

Symptoms of Serofibrinous Pleurisy.—The disease usually sets in with chilliness, pain, fever, and cough. The pain is stabbing, localized and increased by deep breathing and coughing. The fever ranges, as a rule, between 101° and 103° F., and disappears in from a week to ten days, generally by lysis. The cough is usually hard and dry. The respirations are rapid and shallow, at first on account of the pain, later, as a result of the effusion. Dyspnea may be pronounced if the effusion accumulates rapidly.

In some cases (*latent pleurisy*) the disease begins insidiously, and the presence of effusion is only disclosed by careful physical examination.

PHYSICAL SIGNS.—*Inspection*.—In the first stage there may be deficient expansion on the affected side, owing to the severe pain. After the development of a liquid effusion the character-

istic features are immobility, bulging of the intercostal spaces, and displacement of the apex-beat of the heart.

Palpation reveals immobility of the affected side and absence of the vocal fremitus.

Percussion yields marked dulness or flatness and a sensation of increased resistance. The upper line of dulness is not horizontal, but is curved and rises higher posteriorly. In moderate effusions the level of dulness often changes with the position of the patient. Above the effusion percussion gives a tympanitic note (Skoda's resonance). With the patient in the erect posture a triangular patch of dulness can usually be detected along the spine at the base of the unaffected side (*Grocco's sign*). In left-sided effusions Traube's semilunar space is obliterated.

Auscultation.—In the early stage this detects a to-and-fro friction-sound of respiratory rhythm. After the development of the effusion the respiratory sounds are weak and distant. Occasionally they have a tubular quality, especially near the margin of the liquid. Vocal resonance is usually diminished or absent, but occasionally, if the effusion is moderate, egophony may be heard. The friction-sound may again be audible when the fluid disappears.

Mensuration shows an increase in the size ($\frac{1}{2}$ to 1 inch) of the affected side.

Cytodiagnosis.—A preponderance of lymphocytes in the exudate suggests chronic tuberculous pleurisy, while a great excess of polynuclear leukocytes points to pneumococcic or streptococcic infection.

Diagnosis.—*Lobar Pneumonia*.—The severe chill, rusty expectoration, high fever, the fine inspiratory râles, dulness not changing with the patient's posture, increased vocal fremitus, increased vocal resonance, loud bronchial breathing, and the absence of bulging and of displaced apex-beat will serve to distinguish pneumonia from pleurisy.

Pleurodynia.—In this affection the pain and tenderness are diffuse; moreover, fever, friction-sounds, and signs of effusion are absent.

Diaphragmatic Pleurisy.—This may present the following symptoms: Intense pain under the margin of the ribs, with tenderness on pressure; thoracic breathing; tenderness over the phrenic nerve, which is accessible between the two roots of the sternocleidomastoid at the base of the neck; hiccup; and severe dyspnea. The physical signs are not marked.

Pericarditis with Effusion.—In this condition the area of dulness has a characteristic shape, the sounds of the heart are distant and muffled, and there is greater embarrassment of the circulation.

Hydrothorax.—In this condition pain and fever are absent. There is often a history of cardiac or renal disease, and the fluid on aspiration is found to contain less than 3 per cent. of albumin and to have a specific gravity below 1015.

Pyothorax.—This may be recognized by the general symptoms of sepsis—persistent irregular fever, increasing pallor, profuse sweats, chills, and leukocytosis. In doubtful cases it will be necessary to resort to the exploratory needle.

Prognosis.—In simple serofibrinous pleurisy the prognosis is guardedly favorable. Fever usually subsides in from a week to ten days, and absorption of the fluid in many cases is complete in from four to six weeks. In other cases (usually tuberculous) the effusion remains for months or recurs again and again after aspiration. Sudden death occasionally occurs when the fluid is excessive. In about one-fourth of the cases signs of pulmonary tuberculosis sooner or later develop.

Treatment.—The patient should be kept in bed and restricted to a liquid or semiliquid diet. Mercurial or saline aperients may be prescribed at the onset. For the severe pain the application of a blister or of wet or dry cups, together with the administration of morphin, will be found effective. Strapping the affected side with broad strips of adhesive plaster is also useful. Acute sthenic cases with decided fever are often favorably influenced by the administration of salicylates (1 to 1½ drams of the sodium or ammonium salt a day). In asthenic cases salicylates are of no avail.

Removal of Serous Effusion.—The most useful measures for promoting absorption are the application of iodine or of flying blisters, and the administration of hydragogue cathartics and of diuretics. From $\frac{1}{2}$ to 1 ounce of magnesium sulphate may be given in as little water as possible an hour before breakfast, and the fluid consumed by the patient during the day restricted to a minimum. The most serviceable diuretics are caffeine, theobromine, and potassium citrate or acetate. Potassium iodide (5 to 10 grains thrice daily) is also employed for its absorbent effect. Diaphoretics are of no value.

Paracentesis is demanded—(1) if the effusion is considerable and shows no signs of receding after the lapse of two weeks; (2) if there is sufficient fluid to cause severe dyspnea, cyanosis, persistent cough, or failing pulse; (3) if the fluid reaches the level of the third rib and there is marked dislocation of the adjacent organs; (4) if the presence of pus is suspected.

The most favorable site for the puncture is usually in the sixth or seventh intercostal space, between the mid-axillary line and the angle of the scapula. After anesthetizing the part, the needle should be introduced with a quick stroke along the upper margin of the rib. The fluid should be removed slowly, and under no circumstance should extreme efforts be made to obtain the largest possible amount. The operation should be terminated at once if incessant cough, severe pain, dyspnea, palpitation, tendency to syncope, or other untoward symptoms appear.

Purulent Pleurisy (Empyema, Pyothorax).—The effusion may be primarily purulent, having been excited by pyogenic microorganisms, or a serofibrinous effusion, through subsequent infection, may become purulent. Traumatism or the rupture of a purulent accumulation into the pleural sac is an occasional cause. It frequently follows pneumonia, particularly in children, in whom the most common form of pleurisy is empyema. It is sometimes secondary to tuberculosis or one of the infectious fevers.

The organisms most frequently present are the pneumococcus, streptococcus, staphylococcus, and tubercle bacillus.

Symptoms.—The physical signs and symptoms are similar to those observed in serofibrinous pleurisy. Pus is indicated by septic phenomena—high and irregular fever, sweats, chills, pallor, and leukocytosis; by the results of aspiration; and sometimes by edema of the chest-walls. In pulsating pleurisy the effusion is almost always purulent.

Prognosis.—Grave, though recovery frequently occurs. The most favorable cases are those following pneumonia.

Treatment.—This consists in free incision and thorough drainage. Irrigation is unnecessary unless the fluid is putrid. In long-standing cases the excision of several ribs (Estlander's operation) facilitates retraction and the obliteration of the pleural sac, which is essential to a cure.

HYDROTHORAX

Definition.—An accumulation of serum of non-inflammatory origin in the pleural sac. The condition may be unilateral or bilateral.

Etiology.—It may result from the usual causes of edema—nephritis, chronic heart disease, emphysema, anemia, etc., it may be due to pressure on the azygos or pulmonary veins by a tumor, aneurysm, or dilated right auricle. In cardiac disease it is usually unilateral and on the right side, or if bilateral more marked on the right side.

Symptoms.—It gives rise to dyspnea, cyanosis, and the physical signs of a pleural effusion.

Diagnosis.—This is based upon the history, the absence of pain and fever, and the character of fluid obtained by aspiration (see p. 296).

Treatment.—Remedies should be directed to the primary disease. If hydragogue cathartics and diuretics fail to afford relief and the dyspnea becomes urgent, aspiration must be practiced.

PNEUMOTHORAX

Definition.—The presence of air or gas in the pleural sac.

Etiology.—About 85 per cent. of the cases result from the rupture of a tuberculous cavity into the pleura. Less common causes are rupture of the lung from abscess, gangrene, emphysema, etc.; rupture into the lung of an empyema; penetrating wounds of the chest (including thoracentesis); and perforation of the pleura in ulcer or cancer of the esophagus or stomach. Very rarely gas is produced spontaneously in a pleuritic exudate by the gas bacillus (*Bacillus aërogenes capsulatus*).

Pathology.—The adjacent viscera are often much displaced and the lung is compressed. Even when air alone has escaped into the pleural sac, an effusion soon forms, so that *pneumohydrothorax* or *pneumopyothorax* is an almost inevitable result.

Symptoms.—The onset is frequently characterized by localized pain, marked dyspnea, cough, fall in temperature, feeble pulse, and even a condition of collapse. If, however, the air is confined by adhesions or effusion to a small area (circumscribed pneumothorax) no special symptoms may arise.

PHYSICAL SIGNS.—*Inspection* may reveal distention of the affected side, immobility, and marked displacement of the apex-beat of the heart.

Palpation.—Vocal fremitus is diminished or absent.

Percussion.—The note is usually tympanitic, but it may be dull if the sac is very tense. At the base there may be flatness, changing with the posture of the patient.

Auscultation.—The respiratory murmur and vocal resonance are usually absent, but if the opening in the lung remains patulous, amphoric breathing may be detected. Metallic tinkling is often heard. When a silver coin is placed on the affected side and is struck with another, the auscultator may detect a clear metallic sound (bell-tympany). If fluid is present, shaking the patient elicits a splashing sound (Hippocratic succussion).

Diagnosis.—*A Large Tuberculous Cavity.*—This is usually located near the apex instead of the base; the surface is sunken, not prominent; the heart is not displaced; succussion-splash and bell-tympany are rarely obtainable.

Dilated Stomach.—This may give a tympanitic note over the left pulmonary base and may simulate a pneumothorax; but the tympanitic note is continued down into the abdomen, and the swallowing of liquid is distinctly audible over the base of the chest.

Prognosis.—This depends on the cause. In tuberculous subjects it usually proves fatal in from a few days to a few months. In empyemic and traumatic cases the outlook is distinctly more favorable.

Treatment.—In tuberculous cases the indications are to relieve distress by morphin and to combat collapse by such stimulants as ether, ammonia, camphor, alcohol, and strychnin. Aspiration occasionally affords temporary relief. In non-tuberculous cases of pneumopyothorax operative intervention is usually advisable.

HEMOTHORAX

Definition.—Blood in the pleural cavity.

Etiology.—It usually results from wounds of the chest-wall, fracture of the ribs, or the rupture of an aneurysm. A sanguineous inflammatory (*hemorrhagic pleurisy*) exudate frequently occurs in cancerous and tuberculous pleurisy and in simple pleurisy if the individual is profoundly anemic.

Symptoms.—The symptoms and physical signs are those of pleural effusion.

INFECTIOUS DISEASES

FEVER

FEVER is an abnormal condition, characterized by elevated temperature, quickened respiration and circulation, faulty secretions, and increased tissue-waste. It is dependent upon a perversion of the physiologic processes whereby the generation and the loss of heat are so balanced as to maintain the normal temperature.

The Detection of Fever.—There is only one reliable way of detecting fever, and that is by means of the clinical thermometer. The instrument may be placed in the mouth, axilla, rectum, or vagina.

When the mouth is selected, the bulb should be placed under the tongue and the lips kept closed. Hot or cold drinks recently taken mar the result. For obvious reasons the mouth should not be used in delirious patients.

When the axilla is selected, the following precautions must be observed: Wipe off the perspiration and dry the skin; insert the bulb of the instrument deep in the armpit, and see that the arm is kept close to the side. The thermometer should be kept in position until the mercury maintains the same level for two minutes; this will usually require in all about six or seven minutes.

The rectum may be selected in children. The rectal temperature is slightly above that of the mouth and from $\frac{1}{2}^{\circ}$ to 1° F. above that of the axilla.

Febrile Stages.—The course of all fevers is marked by three stages: (1) Invasion; (2) fastigium, or stadium; (3) deferescence, or decline.

Invasion.—During this period the temperature gradually rises until it reaches its maximum.

Fastigium.—In this period, though there may be marked variations, the temperature shows a tendency to touch again and again its highest point.

Defervescence.—In this period the temperature gradually falls until it reaches the norm.

Terminations of Fever.—Fever terminates by lysis or crisis.

Lysis.—The temperature falls slowly by slight gradations until it reaches the norm.

Crisis.—The temperature falls suddenly—often four or five degrees in twelve or twenty-four hours. In croupous pneumonia, typhus fever, erysipelas, relapsing fever, and malarial fever the fever usually ends by crisis.

The Degree of Pyrexia.—The following is Wunderlich's classification of febrile temperatures:

1. Subfebrile, temperature 99.5° – 100.4° F.
2. Slightly febrile, temperature 100.4° – 101.3° F.
3. Moderately febrile, temperature 101.3° – 103.1° F.
4. Decidedly febrile, temperature 103.1° – 104° F.
5. Highly febrile, temperature above 103.1° F. in the morning and above 104.9° F. in the evening.
6. Hyperpyretic, temperature above 106° F.

Febrile Remissions.—All fevers show diurnal variations. The maximum temperature is usually reached at about 6 P. M. and the minimum at about 6 A. M. Occasionally these extremes are reversed and the maximum is in the morning and the minimum in the evening. The daily difference usually amounts to about 1° F.

Types of Fever.—According to the degree of the diurnal variation three types are recognized:

1. *Continued Fever.*—The diurnal variation is slight— 1° – 1.5° F. Typhus fever, pneumonia, and scarlet fever are examples of continued fevers.

2. *Remittent Fever.*—The diurnal variation is marked, but the minimum temperature is still above the norm. Typhoid

fever, remittent fever, and septic fever are examples of this type.

3. *Intermittent or Interrupted Fever*.—The diurnal variation is marked, and the minimum is normal or subnormal. The following fevers show multiple intermissions:

1. Intermittent malarial fever.
2. Relapsing fever.
3. Malta fever (the febrile periods usually last from two to three weeks and the afebrile periods, a few days).
4. Septicemic fever (this may be intermittent or remittent).
5. Hepatic intermittent fever (see p. 101).

A *single intermission or marked remission* is observed in the following fevers:

Smallpox (a remission occurs about the third day).

Yellow fever (a remission usually occurs about the third or fourth day).

Measles (a distinct remission often occurs on the second or third day).

Dengue (a decided remission occurs on the third or fourth day and lasts about a day).

Causes of Fever.—The chief cause is disturbance of the heart-regulating centers by toxic substances circulating in the blood. These substances may owe their origin to bacterial invasion (acute infectious diseases), to faulty metabolism (acute gout, thermic fever), or to mechanical, thermic, or chemical injury of the tissues. Occasionally, as in hysteria, fever appears to be due to a direct disturbance of the heat-regulating centers.

Symptoms of Fever.—The temperature is elevated, the pulse is accelerated, the respirations are increased, the tongue is coated, the appetite is impaired, the secretions of the alimentary canal are deficient, and the urine is scanty, dark colored, and of high specific gravity. Persistent fever is attended with more or less wasting of the body.

The pulse-temperature ratio:

A temperature of 98.4° F. corresponds to a pulse of 70.

A temperature of 100° F. corresponds to a pulse of 80–90.

A temperature of 102° F. corresponds to a pulse of 100-110.
A temperature of 104° F. corresponds to a pulse of 120-130.

Effects of Fever on the Tissues.—High and long-continued fever induces certain marked changes in the tissues, especially cloudy swelling, fatty degeneration, and coagulation necrosis.

Period of Incubation.—The period elapsing between the occurrence of the infection and the development of symptoms.

It varies considerably in the same disease, being more or less influenced by the susceptibility of the patient and the virulence of the contagion. The average period of incubation in the various fevers is as follows:

Typhoid fever: five to twenty-one days.

Typhus fever: a few days to two weeks.

Measles: seven days to two weeks.

Rötheln or rubella: five days to three weeks.

Scarlatina: three to seven days.

Smallpox: ten days to two weeks.

Erysipelas: three to seven days.

Diphtheria: two to seven days.

Varicella: fourteen to sixteen days.

Tetanus: a few days to three weeks.

Mumps: two to three weeks.

Yellow fever: from two to five days.

Cholera: two to five days.

The date at which rashes appear in the various acute infections:

Typhoid fever: seventh to the ninth day.

Typhus fever: fourth or fifth day.

Smallpox: third or fourth day.

Measles: third or fourth day.

Scarlatina: first or second day.

Rötheln or rubella: first or second day.

Varicella: first day.

Protection from Future Attacks.—Few diseases confer absolute immunity against future attacks, but the following are fairly protective:

Typhoid fever: relapses are common, but second attacks are infrequent.

Typhus fever: second attacks are very rare.

Measles: second attacks are very rare.

Rubella: second attacks are rare.

Scarlet fever: second attacks are rare.

Smallpox: second attacks occasionally occur.

Mumps: second attacks are rare.

Varicella: second attacks are uncommon.

Yellow fever: second attacks are rare.

Cerebrospinal fever: second attacks have been occasionally observed.

The following specific fevers do not confer immunity:

Erysipelas.

Malarial fever.

Relapsing fever.

Influenza.

Diphtheria.

Lobar pneumonia.

Rheumatic fever.

Cases of Protracted Fever.—Fever of a remittent or intermittent type lasting from a few weeks to two months or longer, and unaccompanied by very definite physical signs may occur in: (1) Typhoid fever; (2) malarial fever (estivo-autumnal infection); (3) tuberculosis; (4) septicemia (in particular subacute or chronic infectious endocarditis); (5) infection of the urinary passages by the colon bacillus; (6) certain diseases of the blood or hemopoietic system, such as pernicious anemia, leukemia and Hodgkin's disease; (7) secondary and tertiary syphilis; (8) chronic cerebrospinal fever; (9) Malta fever; (10) kala-azar; (11) trypanosomiasis.

Infections in which jaundice is likely to occur:

Yellow fever.

Relapsing fever.

Certain types of malaria (bilious remittent fever and black-water fever).

Spirochetosis icterohemorrhagica (Weil's disease).

SUBNORMAL TEMPERATURE

Temperatures below 97.5° F. may be considered subnormal. They are observed in the following conditions:

1. During convalescence from certain febrile diseases. After pneumonia and typhoid fever the temperature may remain subnormal for several days.
2. In collapse from various causes.
3. In cholera. In this disease the temperature may be very low (90°–85° F.) for several days.
4. In certain chronic diseases, especially myxedema, diabetes, carcinoma and chronic cardiac disease.

FEBRICULA

(Simple Continued Fever; Ephemeral Fever)

Definition.—An acute febrile disease, of short duration, without definite lesions or a specific etiology.

Etiology.—It is generally met with in young and sensitive individuals. Exposure to the sun, prolonged physical or emotional excitement, and errors in diet seem to excite it.

Symptoms.—The disease usually begins abruptly with chilliness, headache, malaise, and fever which soon attains a maximum of 102° or 103° F. The face is flushed; the pulse is full and rapid; the urine is scanty and high colored; the tongue is coated; the appetite is lost; and the bowels are constipated. There is no characteristic eruption, but herpes is frequently observed on the lips.

The disease lasts from a few days to two weeks, and may end by crisis or lysis.

Diagnosis.—Care must be taken to exclude local inflammations, such as tonsillitis and otitis media, and other fevers.

Typhoid Fever.—At first the diagnosis may be impossible, but the absence of diarrhea, tympanites, abdominal tender-

ness, splenic enlargement, Widal reaction, and eruption will soon make the diagnosis apparent.

Estivo-autumnal Fever.—The history, the splenic enlargement, and the presence of hematozoa in the blood will serve to distinguish this disease from simple continued fever.

Prognosis.—Favorable.

Treatment.—The patient should be confined to bed and placed upon a liquid or semiliquid diet. Fractional doses of calomel may be employed to relieve constipation.

The fever is best controlled by sponging with water and alcohol and by the use of some mild refrigerant mixture like the following:

R. Tincturæ aconiti..... ℥xxiv
 Spiritus ætheris nitrosi..... f℥v
 Liquoris ammonii acetatis..... q. s. ad f℥iij.—M.
 Sig.—A dessertspoonful every two hours for a child of four years.

TYPHOID FEVER

(Enteric Fever; Typhus Abdominalis)

Definition.—An acute infectious disease, excited by the *Bacillus typhosus*, characterized anatomically by definite lesions in Peyer's patches, mesenteric glands, and spleen; and manifested clinically by fever, headache, delirium, abdominal distention and tenderness, diarrhea, enlargement of the spleen and a rose-colored rash.

Etiology.—Typhoid fever occurs at all ages, but it is especially frequent in the second and third decades. It appears in all seasons, but it is most prevalent in the late summer and in autumn. Certain persons and certain families seem to be more liable to infection than others. One attack usually, but not invariably, confers permanent immunity.

The feces and urine of the patient are the sources of infection, and drinking water contaminated with them is the chief medium of transmission. Milk contaminated after leaving the cow is another important medium of infection. Many cases have been traced to articles of food eaten raw, such as

oysters fattened in befouled water, or celery, lettuce, etc., grown in polluted soil. Flies may be an important agent in disseminating the disease. Persons closely associated with typhoid fever patients, such as nurses and attendants, are sometimes infected directly by the stools or urine. Small outbreaks are often traceable to so-called "carriers," persons who though perfectly healthy constantly have typhoid bacilli in their stools. It is probable that in rare instances infection occurs by way of the respiratory tract.

Pathology.—The characteristic lesions are found in the abdominal lymphatics, namely, in Peyer's patches, solitary glands, and mesenteric glands. The changes in Peyer's glands are best studied in the lower part of the ileum, which should be opened on the side of the mesenteric attachment.

In the first few days the glands are swollen and hyperemic; later there is a marked cell-proliferation, the blood-vessels are compressed, and the glands become pale and prominent (medullary infiltration). If the disease advances, necrosis sets in, the glands becoming yellow and soft. In a few days the necrotic tissue is discharged, leaving an oval, ulcerated surface with somewhat irregular margins, and a smooth base formed by the submucous coat, muscular coat, or peritoneum. In the fourth week cicatrization begins, and the gland is ultimately replaced by a smooth depressed scar.

In mild cases the stage of ulceration may not be reached, the proliferated cells being removed by fatty degeneration and absorption without rupture of the gland. The solitary and mesenteric glands pass through similar changes, but the latter rarely rupture. Other lesions are found that are not characteristic. The mucous membrane of both the small and large intestines shows catarrhal changes. The spleen is soft and swollen. The liver, kidneys, and heart reveal parenchymatous degeneration. The respiratory tract is commonly the seat of catarrhal inflammation.

In rare instances general infection occurs without intestinal lesions (*typhoid bacteremia* or *septicemia*).

Symptoms.—The *period of incubation* is from one to three weeks. In this period the patient's health may be unaffected, but as a rule, there are certain *prodromal symptoms*, such as malaise, chilliness, headache and backache, nose-bleed, anorexia, diarrhea, and cough.

The Attack.—*Fever.*—The temperature rises gradually, reaching its maximum (104° – 105° F.) by the end of the first week; it remains at this elevation for another period of from ten days to two weeks, when a gradual defervescence begins and occupies a third period usually lasting about a week. Throughout its course the fever is characterized by marked daily remissions, the evening temperature being from one to three degrees higher than the morning temperature.

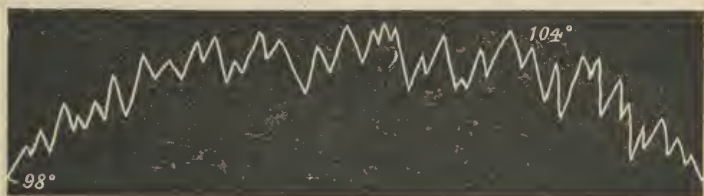


FIG. 11.—Temperature-curve in typhoid fever.

In some cases, especially in the young, the temperature rises quite abruptly. Slight diurnal remissions usually indicate a protracted case. As defervescence advances the temperature becomes more irregular, the remissions are more decided, and occasionally the higher temperature is recorded in the morning. An abrupt fall of several degrees should suggest intestinal hemorrhage or perforation.

Respiratory Symptoms.—These include hurried breathing, cough with tenacious sputum, and numerous bronchial râles.

Circulatory Symptoms.—As the disease progresses the pulse becomes frequent (90–130), weak, and dicrotic. In the early stages the rate is usually less than would be expected from the fever. At the height of the disease the systolic pressure aver-

ages about 110 mm. The heart-sounds become feeble. The first may be especially weak, and resemble the second.

The Face.—The expression is dull and heavy, the cheeks are somewhat flushed, the conjunctivæ are clear, and the pupils often slightly dilated.

The tongue is tremulous; at first it is red at the tip and edges, and covered posteriorly with a whitish fur. In severe cases the tongue becomes dry, brown, and fissured, and *sordes* collect on the teeth.

The Stomach.—Gastric symptoms are not common, but obstinate vomiting sometimes develops and becomes a serious complication.

Intestinal Symptoms.—The abdomen is usually distended. Tenderness is frequently noted on palpation; it may be general or confined to the right iliac fossa. Gurgling may also be detected in the latter region, but it has little significance. Diarrhea is usually present, though it is not a constant symptom. The discharges vary in number from three to six or more a day; they are thin, offensive, and of a yellowish color (likened to pea-soup); on standing, a turbid liquid rises to the top and a granular sediment falls to the bottom. Intestinal hemorrhage occurs in from 5 to 7 per cent. of the cases.

The Eruption.—This appears from the seventh to the ninth day, and is most abundant on the abdomen, though it is not infrequently observed on the chest and back. It is composed of small, slightly elevated rose-colored spots that disappear on pressure. It comes out in successive crops over several days. It may be absent, particularly in the aged and very young. Rarely, in malignant cases, the eruption is petechial. Sudamina and miliaria are common. Herpes is rare.

Splenic enlargement is rarely absent. Rupture has occurred in a few instances.

Nervous Symptoms.—In mild cases apathy, headache, and slight deafness may be the only nervous symptoms. In severe cases there may be muttering delirium, stupor, twitching of

the tendons (subsultus tendinum), picking at the bed-clothes or imaginary objects (carphologia), and ultimately coma.

The Blood.—The number of leukocytes is diminished (5000–3000). In the later stages the hemoglobin and the number of red cells are also more or less reduced. The blood-serum agglutinates the typhoid bacillus (*Widal reaction*). The reaction may be obtained at the end of the first week, and is to be considered positive if there is general clumping of the bacilli with a dilution of 1 to 50 in one hour. It may persist for months or years after recovery. The typhoid bacillus may often be grown from the blood before the Widal reaction is given.

The urine is usually scanty and slightly albuminous. In many cases (25 per cent.) it contains typhoid bacilli. Retention is common.

Convalescence is marked by anemia, falling of the hair, desquamation of the cuticle, and sometimes by mental enfeeblement.

Varieties.—*Mild Typhoid.*—There is moderate fever with marked remissions; the diarrhea is slight; nervous symptoms are often absent; the rash is usually present, and may be abundant.

Abortive Typhoid.—There is an abrupt onset with severe symptoms, but convalescence follows in from ten days to two weeks.

Walking Typhoid.—The symptoms are mild and often disregarded by the patient, who refuses to go to bed; but grave symptoms may develop suddenly, and death from perforation is not uncommon.

Typhoid in Children.—The rash is often absent; the temperature rises abruptly; cerebral symptoms are frequently marked; hemorrhage is comparatively rare; the course is usually mild.

Complications.—Any symptom aggravated constitutes a complication; thus high fever, excessive diarrhea, and tympanites may be troublesome complications.

Hemorrhage.—This usually occurs during the third week, and is indicated by a sudden fall of temperature, followed by dark-red or tarry stools. After large hemorrhages there may be symptoms of collapse.

Perforation.—This occurs in 2 to 3 per cent. of all cases and in from 10 to 20 per cent. of the fatal cases. The suggestive symptoms are localized pain and tenderness, fall of temperature, leukocytosis, a pinched expression, vomiting, marked tympanites, disappearance of the liver dulness, rigidity of abdominal muscles, vesical irritation, and signs of peritonitis.

Laryngitis, lobar pneumonia, hypostatic congestion of the lungs are common complications.

Among the less frequent complications or sequelæ may be mentioned: cholecystitis (not rarely leading to cholelithiasis), phlebitis, peripheral neuritis, pyelitis, nephritis, otitis media, furunculosis, parotitis, periostitis, spondylitis (typhoid spine), and posttyphoidal insanity (rarely permanent).

With the exception of myocarditis, cardiac complications are rare.

Relapse and Recrudescence.—*Relapses* are common (about 10 per cent. of all cases); they repeat the symptoms of the original attack, but are generally milder and of shorter duration, and seldom prove fatal.

Recrudescence.—This is a sudden temporary elevation of temperature occurring during convalescence, and is not associated with a return of the other symptoms. It is usually due to constipation, excitement, or irritating food.

Diagnosis.—*Acute miliary tuberculosis* often closely resembles typhoid fever. In tuberculosis the temperature is usually more irregular; the abdominal symptoms are less marked; pulmonary symptoms, especially dyspnea, are more marked; the rash is absent; the Widal reaction is absent; tubercles may occasionally be detected on the retina; and symptoms of basilar meningitis may be present, such as inequality of the pupils, ptosis, and strabismus.

Malignant Endocarditis.—The following features would suggest endocarditis: The history of a primary disease which might induce endocarditis; irregular fever; intercurrent rigors; moderate leukocytosis; precordial pain and endocardial murmurs; embolic lesions; and the absence of a rose-colored rash, of the Widal reaction, and of marked abdominal symptoms.

Enteritis.—The absence of high fever, of eruption, of splenic enlargement, of epistaxis, and of bronchial catarrh will serve to distinguish enteritis from typhoid fever.

Meningitis.—The abrupt onset, the early development of cerebral symptoms, the irregular fever, the leukocytosis, and the absence of the characteristic rash, of abdominal symptoms, and the Widal reaction will indicate meningitis.

Prognosis.—The prognosis is always doubtful. The mortality varies in different outbreaks of the disease, but the average is about 8 per cent.

A pulse-rate persistently above 120, excessive diarrhea, marked tympanites, severe nervous symptoms, hypostatic congestion of the lungs, and intestinal hemorrhage are unfavorable features.

Prophylactic Measures.—These comprise the obtaining of a pure water supply, the proper disposal of excreta, precautions against food (especially milk) contamination, protection against flies, the isolation of the sick, the thorough disinfection of the patient's discharges (feces and urine), and all articles (bed-linen, bed-pans, urinals, etc.) likely to be soiled by the patient, and constant care on the part of those who handle the sick to protect themselves and others against direct infection. During the prevalence of epidemics all drinking water and milk should be boiled and no vegetables or shellfish should be eaten raw unless their source is known to be above suspicion. Typhoid vaccination usually affords protection for two or three years, and is to be strongly recommended in the cases of soldiers, nurses, orderlies and all others who are in special danger of contracting the disease. The vaccine is given hypodermically once a week for three weeks,

the first dose consisting of 500 million killed bacteria, the second, 1000 million, and the third, 2000 million.

Treatment.—As soon as the nature of the disease is recognized the patient should be confined to bed. The room should be large and airy, and provided with efficient means of securing thorough ventilation. The temperature of the room should be maintained between 65° and 70° F. The bed-pan must be used from the beginning until convalescence is well advanced. The stools and urine should be rendered innocuous before being thrown out. This may be done by treating the evacuation with twice its volume of a 5 per cent. solution of chlorinated lime or a 5 per cent. solution of carbolic acid, and allowing it to stand in a covered vessel for two hours before emptying it into the closet. Soiled clothing should be thoroughly boiled.

The diet should be liquid or semisolid, unirritating, and easily digestible. Milk alone (6 ounces every four hours) does not supply the required number of calories (2500–3000), but, as a rule, it should form a large part of the diet. It may be given diluted with lime water, or as buttermilk, malted milk, koumiss, junket, or ice cream. Among other suitable foods may be mentioned raw or soft boiled eggs, chicken jelly, milk toast, strained oatmeal gruel, potato puree, tea, coffee, cocoa, fruit juices, wine jelly, and custard. Beef tea and broths may be harmful. In the event of digestive disturbances the diet should be restricted for a time to whey or albumin-water. Water, plain or flavored (lemonade, soda-water, etc.), should be given in large amounts between the feedings. When the first sound of the heart becomes weak and the pulse dicrotic, alcohol is usually indicated. From 4 to 6 ounces of whisky or brandy may be given in the twenty-four hours, the amount being determined by the general effect.

The cold bath or the cold pack affords the best means of controlling fever and preventing the development of severe nervous symptoms. It may be employed every three or four hours when the temperature is 102.5° F. or over. Hemorrhage,

signs of perforation, menstruation, and great prostration are contraindications.

Heart-failure.—Cold bathing and the timely use of alcohol do much to guard against heart-failure. When the tendency to cardiac failure is pronounced, strychnin may be given in doses of from $\frac{1}{40}$ to $\frac{1}{20}$ grain every three or four hours. In severe cases the drug should be given hypodermically. Digitalis may also be tried, but in the presence of fever it often proves ineffectual. Salt solution (500 c.c. to 750 c.c. once or twice a day, by the bowel or subcutaneously) is often of service. If collapse is threatened, ether, alcohol, or, better still, camphor (1 to 2 grains in sterile olive oil) may be given subcutaneously every two or three hours.

Diarrhea.—It is advisable to substitute albumin-water for all other food and to give bismuth subnitrate (20 gr.) with a mild antiseptic and codein by the mouth. If the diarrhea be troublesome, silver nitrate may be given by the mouth in combination with opium:

℞. Codeinæ sulphatis..... gr. ii-iv
 Beta-naphtholis..... gr. xl
 Bismuthi subnitratis..... ʒss.—M.

Fiant chartulæ No. xii.

SIG.—One every three or four hours.

℞. Argenti nitratis..... gr. iij
 Pulveris opii..... gr. vj.—M.

Fiant pilulæ xii.

SIG.—One pill every three or four hours.

In very obstinate cases copper sulphate with opium in pill sometimes proves efficacious.

Constipation.—This may be relieved by enemas of soap and water or by the administration of fractional doses of calomel.

Tympanites.—This may often be relieved by reducing the diet to albumin-water, applying turpentine stupes, giving by the mouth small doses of creosote, turpentine, or betanaphthol, and using enemas containing asafetida or turpentine. Enteroclysis is sometimes useful. If extreme, a soft rectal

tube may be introduced into the bowel. Physostigmin sulphate ($\frac{1}{50}$ grain twice a day) is of service.

Hemorrhage.—Absolute rest is imperative. Cold bathing should be suspended. It is advisable to elevate the foot of the bed. An ice-bag may be applied with advantage to the right iliac region, and ice may be given to suck. The best drug is morphin ($\frac{1}{6}$ to $\frac{1}{4}$ grain) hypodermically. Ergot is useless. In cases of recurrent hemorrhage blood-serum (20–30 c.c.) may be given subcutaneously and gelatin by the mouth.

Insomnia.—Morphin is usually the best somnifacient. In some cases, however, sodium bromid, barbital, or chloralamid acts better.

Delirium is best managed by hydrotherapy. Low, muttering delirium usually calls for alcoholic stimulation. An ice-cap is useful. Salt solution by the bowel, or in serious cases subcutaneously, is often serviceable. Camphor or musk may be tried: the former is best given hypodermically, the latter, by the bowel. In active or violent delirium no drug is so generally useful as morphin.

Retention of Urine and Bacilluria.—Retention of urine, a common symptom, is sometimes overcome by the use of hot applications or an enema of hot water. In many cases, however, it is necessary to use the catheter. For the bacilluria, hexamethylenamin (5 to 10 grains thrice daily) is the best remedy.

Perforation.—Recovery from peritonitis is so exceedingly rare under medical treatment that operative intervention is called for in all cases that are not obviously moribund. The operation should be done at the earliest possible moment. Keen has collected 83 cases with 16 recoveries.

LOBAR PNEUMONIA

(Croupous Pneumonia)

Definition.—An acute infectious disease with a pulmonary localization, characterized clinically in its ordinary form by

high temperature, cough, dyspnea, blood-tinged expectoration, variable toxemia, and physical signs indicating consolidation of one or more lobes of the lungs.

Etiology.—The disease occurs at all ages. It is probably most common in early adult life. Males are attacked more frequently than females. The majority of cases occur in the winter and spring months. Lowered vitality from pre-existing disease (typhoid fever, influenza, nephritis, arteriosclerosis, diabetes, etc.) or excessive fatigue, exposure to cold, and alcoholism are important predisposing factors. Traumatism has a slight etiologic influence. One attack seems to render the patient more liable to subsequent infection.

The exciting cause of typical croupous pneumonia in the vast majority of cases is the *Diplococcus pneumoniae* (pneumococcus) of Fränkel, a lance-shaped coccus, usually growing in pairs. Other organisms are rarely present in pure culture. Four strains of pneumococci may be recognized by agglutination or immunity reactions (Types I, II, III, and IV). Types I and II are the cause of most of the cases of pneumonia; Type III has the highest pathogenic power; and Type IV, which is often present in the normal human mouth, has the lowest pathogenic power.

Pathology.—Anatomically three stages have been recognized: (1) That of congestion; (2) that of red hepatization; (3) that of gray hepatization.

Stage 1.—The affected portion remains distended when the chest is opened; it is of a deep-red color, and is more resistant to the touch than the normal lung. On section, a frothy, blood-stained serum freely exudes. Microscopic examination reveals a dilated and tortuous condition of the capillaries, swelling of the alveolar cells, and a slight corpuscular exudate.

Stage 2.—The hepatized portion is increased in volume, is quite firm, is of a dark-red color, and so heavy that it sinks in water. It is very friable, and the torn surface is dry and presents a granular appearance, owing to the projection of fibrinous plugs from the alveoli.

Microscopic examination reveals a mesh of coagulated fibrin inclosing numerous red blood-corpuscles and some leukocytes. The latter are also noted in the interlobular tissue. In sections properly treated the diplococcus may be detected.

Stage 3.—The red color gives place to a mottled gray, and the solidified area begins to soften. The change in color is due to the compression of the capillaries, to the disintegration of red corpuscles and their replacement by polymorphonuclear leukocytes, and to fatty degeneration of some of the elements.

The consolidation usually begins at the base and extends upward. The most frequent seat is the lower lobe of the right lung. The bronchi and the adjacent pleura are involved in the inflammatory process.

Events.—Resolution commonly occurs, the exudate first undergoing liquefaction (autolysis) through the agency of a ferment liberated by the leukocytes and then being rapidly removed by absorption. Death may occur at any period of the disease from general toxemia, the severity of which is often altogether disproportionate to the area of lung involved; from dilatation of the right ventricle; from asphyxia; or a pneumococcic complication, such as meningitis or endocarditis.

Abscess, gangrene, and chronic interstitial pneumonia are rare terminations.

Symptoms.—The disease usually begins with a decided *chill* and a sharp *pain* in the side, followed by a rapid *rise of temperature*. The latter often attains its maximum (104° – 105° F.) in twenty-four hours, and generally continues high, with slight diurnal remissions, for a period from five to ten days, when it falls by crisis, frequently reaching the normal within twenty-four hours. In protracted cases the temperature not rarely falls by lysis. There is marked dyspnea and the respirations are shallow and rapid, sometimes reaching 60 or even 70 per minute. The pulse is accelerated but not in proportion to the respiration, so that the *pulse-respiration ratio* may be 3 to 1 or 2 to 1, instead of 4 to 1. In unfavorable cases the pulse becomes very frequent (140 –

150) and weak. *Cough* is a prominent symptom: at first it is short and dry, but later it is accompanied by *bloody or rusty, translucent and tenacious sputa*. Microscopically, the sputum contains red blood-corpuscles, free pigment, pus-corpuscles, diplococci, and other micro-organisms. The face is flushed; the lips are cyanosed and often the seat of an herpetic eruption; the tongue is heavily furred; the bowels are constipated; the urine is scanty, high-colored, deficient in chlorids, and often slightly albuminous. In severe cases *delirium* is rarely absent. Examination of the blood usually shows marked polynuclear leukocytosis (15,000 to 40,000).

PHYSICAL SIGNS.—*Inspection*.—There may be deficient expansion over the affected side. There is no bulging of the interspaces nor displacement of the apex-beat.

Palpation.—In the vast majority of cases the vocal fremitus is considerably increased over the affected area.

Percussion.—In the earliest stage there may be hyperresonance from diminished intrapulmonary tension. As consolidation advances, however, the note becomes remarkably dull. Percussion over unaffected lobes yields a tympanitic note (Skoda's resonance).

Auscultation.—In the stages of congestion fine crepitant râles are heard at the end of full inspiration. They are probably produced by the forcible separation of adherent vesicular walls. In the stage of consolidation auscultation usually reveals exaggerated vocal resonance and bronchial breathing, but the breath sounds may be absent if the bronchioles are filled with exudation. During resolution the softening of the exudate gives rise to fine moist râles—the *redux crepitus*.

Atypical Cases.—*Senile Pneumonia*.—The symptoms often develop insidiously; the temperature may not be high; the pulse may not be accelerated; expectoration is often absent; the physical signs are not marked; delirium is common; weakness is extreme; and death from exhaustion is the most frequent termination.

Pneumonia in Children.—It is often ushered in with vomiting or convulsions. Headache, delirium, and stupor are prominent symptoms, so that the disease may simulate meningitis. The temperature is very high; expectoration is often absent. The disease frequently begins at the apex of the lung. The duration is usually short.

Typhoid Pneumonia.—In this form there are pronounced typhoid symptoms—headache, muttering delirium, stupor, a dry, brown tongue, subsultus tendinum, carphologia, a rapid, weak pulse, and high fever. The expectoration may resemble prune-juice.

Pneumonia of Drunkards.—The onset is often gradual; the dyspnea is marked; the temperature is not high; maniacal delirium commonly develops; and death from exhaustion is exceedingly frequent.

Massive Pneumonia.—In this form the bronchioles, as well as the air-vesicles, are filled with fibrinous exudate. The physical signs resemble those of pleural effusion.

Central Pneumonia.—In this form the inflammatory process commences in the center of a lobe, and in consequence the characteristic physical signs may not manifest themselves for two or three days.

Migratory Pneumonia.—In this type the specific inflammation shows a tendency to spread and to involve successively fresh areas of lung tissue.

Complications.—These are usually due to pneumococcic infection. Pleurisy is the most common complication. It may be either serous or purulent. Pericarditis and endocarditis are not very infrequent. The latter is sometimes of the malignant type. Among less frequent complications may be mentioned inflammation of the middle ear, meningitis, arthritis, parotitis, nephritis, catarrhal jaundice, acute dilatation of the stomach, and delayed resolution (consolidation may last for five or six weeks and then gradually disappear). Abscess, gangrene, and chronic interstitial pneumonia are rare sequels.

Diagnosis.—*Pleurisy.*—There is rarely a distinct chill; fever is not so high nor the pulse so rapid; there is no rusty sputum; nervous symptoms are wanting; there is often bulging of the interspaces, with displacement of the apex-beat; the level of dulness may change with the posture of the patient; vocal fremitus and vocal resonance are diminished; and the breath-sounds are usually weak and distant.

Acute Pulmonary Tuberculosis.—The history, the mode of onset, the long duration, the remittent fever, the rapid emaciation, profuse sweats, and presence of tubercle bacilli and elastic fibers in the sputum will suggest tuberculosis.

Pulmonary Edema.—In edema there is absence of chill, fever, and pain; the expectoration is frothy and serous; both lungs are commonly affected; auscultation reveals abundant subcrepitant râles and weak breathing.

Typhoid Fever.—Typhoid pneumonia may readily be mistaken for typhoid fever with pneumonia; but pneumonia as a complication occurs late in the disease, so that the history of the onset gives much assistance. Moreover, in typhoid pneumonia there is no roseolar rash or characteristic blood-serum reaction.

Prognosis.—In young, robust subjects of good habits the prognosis is good. After the age of sixty the outlook is grave. In drunkards the disease is especially fatal. The coexistence of heart or kidney disease makes pneumonia exceedingly dangerous.

In any case a pulse more frequent than 120 per minute, especially if weak and irregular, absence of leukocytosis, pronounced cerebral symptoms, and marked meteorism (toxic paresis of the intestine) are unfavorable features. The average mortality is about 20 per cent.

Treatment.—The windows of the sick-room should be wide open, regardless of atmospheric conditions. A temperature below 65° F. is preferable. The diet should be fluid or semi-fluid. Milk, junket, wine-whey, broth, eggs, and gruel are suitable forms of nourishment. Cool water should be given

freely. In the absence of any indication for special local treatment the chest may be enveloped in a light woolen jacket.

In robust subjects, at the onset, when the invasion is violent and attended with a bounding pulse, marked dyspnea, and severe pleuritic pain, the application of several wet cups to the affected area may afford great relief. Later in the course of the disease, if cyanosis and orthopnea develop in consequence of overdistention of the right ventricle, venesection may prove useful.

Specific Treatment.—Homologous serum, if used early, is very effective in pneumonia caused by pneumococci of Type I, which are responsible for about one-third of all cases; but homologous serums in pneumonia caused by other types of pneumococci have not been of much value. Recently, however, encouraging results have been reported from the intravenous injection of a serum-free solution of antibodies of the pneumococci (Huntoon's method), although the treatment results in a severe, sometimes dangerous, reaction.

Circulatory Failure.—Digitalis is the most reliable stimulant. It is advisable to give three or four large doses (30 minims of the tincture) in the first forty-eight hours, so that if need for it arises later in the disease its physiologic effects may be quickly obtained with comparatively small doses. Caffein and strychnin are often effective adjuvants to digitalis. Alcohol, in moderate doses, is useful when there is an alcoholic history; otherwise it is of doubtful value. Atropin is of service when there is excessive bronchial secretion or tendency to pulmonary edema. In acute heart failure camphor (2 grains in sterile olive oil every two hours hypodermically) is worthy of trial. Enteroclysis with saline solution is also useful in combating adynamia.

Pain.—Morphin hypodermically is the best analgesic. Hot or cold applications are useful. When the pain is very severe, a few wet or dry cups will be found serviceable.

Cough.—Hard, dry cough is best relieved by codein ($\frac{1}{8}$ to $\frac{1}{4}$ grain), heroin ($\frac{1}{16}$ grain), or Dover's powder (3 to 5 grains).

Expectorants are rarely needed. If, however, there is much bronchial catarrh, potassium citrate or ammonium chlorid may be given to facilitate expectoration.

Fever.—Very high fever is best controlled by the application of ice-bags to the affected side and by cold spongings. The use of packs or tub-baths is inadvisable.

Dyspnea.—Cardiac and respiratory stimulants (strychnin, caffen, ammonia) are of service. Oxygen makes the breathing easier, lessens cyanosis, and conduces to sleep, and to this extent aids in conserving energy.

Delirium and Insomnia.—An ice-cap to the head is serviceable. In the early stages of the disease morphin is beneficial and safe. Later, preference should be given to bromids, barbital, or chloralamid.

Tympanites.—Turpentine stupes, turpentine ($\frac{1}{2}$ ounce to the pint of water) or asafetida (8 ounces of the official emulsion) enemas, passage of a rectal tube, and proctoclysis with hot saline solution often suffice. In severe cases pituitary extract (15 min. every two or three hours subcutaneously) or physostigmin salicylate ($\frac{1}{60}$ grain subcutaneously, repeated in three hours) should be tried.

Delayed Resolution.—Small blisters may be applied over the affected area, and potassium iodid may be administered internally. X-rays have proved useful in a few cases.

TYPHUS FEVER

(Brill's Disease; Ship Fever; Jail Fever; Tabardillo)

Definition.—An acute infectious disease, unassociated with any characteristic anatomic lesions, and manifested by severe toxemia, a maculopapular eruption, frequently becoming petechial, and high fever lasting about a fortnight and subsiding by crisis.

Etiology.—Typhus fever, formerly widely prevalent and one of the most dreaded of infections, is at present rarely epidemic. It is more or less endemic, however, in England

and Ireland, Russia, southeastern Europe, and Mexico, and small outbreaks occur from time to time in nearly all large cities. Overcrowding and filth are important predisposing factors. Until a few years ago the infection was believed to be air-borne, but the researches of Nicolle, Ricketts and Wilder, and others have demonstrated that the body-louse is the chief agent (probably the essential one) in spreading the disease. The short oval bodies found in the endothelial cells of the small blood-vessels and known as *Rickettsia prowazeki* are probably the exciting agents. One attack of typhus fever usually confers lasting immunity.

Pathology.—There are no characteristic macroscopic lesions. As in other acute infections, the liver and spleen are swollen and the parenchymatous organs show cloudy swelling and fatty changes. The blood is dark and fluid. Microscopically, proliferative changes in the endothelium of the arterial capillaries and a perivascular infiltration of small round cells are constant findings.

Symptoms.—The *period of incubation* is from a few days to two weeks. The disease begins abruptly with a chill, general pains, marked prostration, and fever. The *temperature* reaches its maximum in from two to three days and remains high, with slight morning remissions, until the end of the second week, when it falls by crisis. The pulse is frequent small, and of low tension. The tongue is furred, and in the later stages frequently dry, brown, and fissured. The face is dusky, the conjunctivæ are injected, and the pupils are contracted (“ferret eyes”). As the disease progresses delirium, sometimes maniacal, usually develops, and in grave cases other nervous derangements, such as subsultus tendinum, carphologia, and coma vigil (coma with open staring eyes) supervene. The *rash* appears on the fourth or fifth day. It consists of a single crop of slightly elevated dull-red macules, which frequently become petechial within forty-eight hours. Between the macules there is in many cases a dusky-red mottling (“mulberry rash”). The urine is scanty, high-colored, and

slightly albuminous. Leukocytosis is usually present. In the type studied by Brill nervous symptoms, with the exception of severe headache, are usually absent, and the eruption is rarely petechial.

Complications.—These are not very common, but broncho-pneumonia, hypostatic congestion of the lungs, phlebitis, nephritis, and localized gangrene may occur.

Diagnosis.—*Cerebrospinal Meningitis.*—In this affection the pain in the back is greater; the temperature-curve is much more irregular; there is a marked tendency to opisthotonos and facial palsies; Kernig's sign is present; the spinal fluid

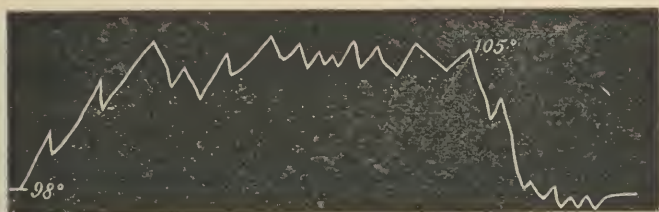


FIG. 12.—Temperature-chart of typhus.

shows characteristic changes; and the eruption, though it may resemble that of typhus, is inconstant and without a special time for appearing.

Typhoid Fever.—The resemblance is in the nervous phenomena only. In typhoid fever the fever rises gradually and subsides by lysis; the eruption appears later, remains rose-red, and does not become petechial; the face is not dusky; the eyes are not injected; the blood yields the Widal reaction, and there are marked abdominal symptoms.

Prognosis.—The mortality varies in different epidemics. It may exceed 50 per cent. or be as low as 2 per cent. Advanced years and alcoholism render the prognosis very grave.

Treatment.—Destruction of body-lice is an important prophylactic measure. The patient should be isolated, his clothing disinfected, and his body sponged with a solution

(1 to 2000) of corrosive sublimate; and the apartment, after his removal, should be thoroughly fumigated with burning sulphur. The general treatment of the disease does not differ from that of typhoid fever.

RELAPSING FEVER

(Spirillum Fever; Famine Fever)

Definition.—An acute infectious disease due to the Spirochæta of Obermeier or closely allied spirochetes, and characterized by recurring paroxysms of high fever lasting for from five to seven days.



FIG. 13.—Temperature-curve in relapsing fever.

Etiology.—This disease prevails at times in Europe, Africa (African tick fever), and India. The last American epidemic was in 1869. It attacks especially young adults, and unsanitary conditions seem to favor its spread. The infection is apparently transmitted by the body-louse and certain species of ticks.

Pathology.—There are no characteristic lesions. The liver and spleen are enlarged, and the latter is frequently the seat of infarctions. The parenchymatous organs show cloudy swelling. There may be hyperplasia of the bone-marrow.

Symptoms.—The *period of incubation* is from five to ten days. The disease begins abruptly with a chill, followed by fever, which reaches its maximum (105° – 106° F.) in twenty-four hours, and remains high for from three to five days, when it falls by crisis. After an intermission of five or six days it

again rises rapidly and remains high for a similar period. Convalescence usually begins at the end of the second paroxysm, but it may not begin until after the third or fourth. Other noteworthy symptoms are intense pains in the head, back, and joints, and the presence of the spirochæta in the peripheral blood during the febrile stage. Gastric irritability and jaundice are not uncommon. Not infrequently there is an ecchymotic rash.

Complications.—These are uncommon, but there may be nephritis, pneumonia, ophthalmia, or hemorrhages from the kidneys, stomach, or intestine.

Diagnosis.—The characteristic febrile paroxysms with the long intermissions and the presence of the spirochæta in the blood are the distinctive features.

Prognosis.—In the absence of jaundice and other complications the prognosis is good. The mortality in different epidemics varies from 3 to 20 per cent.

Treatment.—The prophylactic measures do not differ from those employed against typhus fever (see p. 325). Arsphenamin or neoarsphenamin is virtually a specific. Absolute rest, good nursing, and proper diet will do much to avert complications. Vomiting may be controlled by carbonated water, wine of ipecac (1 minim), diluted hydrocyanic acid (1 or 2 drops), or cocain ($\frac{1}{8}$ grain); the fever by cold sponging; and the severe pains by morphin or acetphenetidin.

CEREBROSPINAL FEVER

(Epidemic Cerebrospinal Meningitis; Spotted Fever)

Definition.—A specific infectious disease, due to the *Diplococcus intracellularis meningitidis*, and characterized by inflammation of the cerebral and spinal meninges.

Etiology.—The disease may be sporadic or epidemic. Overcrowding and other unsanitary conditions predispose to epidemics. Outbreaks are most common in the winter and spring. The young are more susceptible than the aged. The disease is mildly contagious, and meningococcic “carriers”

probably play an important part in its dissemination. Infection is believed to occur through the nose.

The Exciting Cause.—The *Diplococcus intracellularis meningitidis* of Weichselbaum is the specific cause of the disease. This organism appears in the polymorphonuclear leukocytes of the exudate, and may be gotten from the nasal secretion and the fluid obtained by spinal puncture. In the early stages of the disease it may be present also in the blood.

An infection almost identical with cerebrospinal fever may be caused by other organisms, especially the *diplococcus pneumoniae*.

Pathology.—In most cases the membranes of the brain and cord are deeply congested and opaque. Serofibrinous or purulent exudation is found both at the base and on the convexity of the brain, especially in the fissures and along the blood-vessels. The spinal meninges present similar changes, the posterior surface of the cord being particularly involved. The cranial nerves are usually involved and in many cases the ventricles are distended with turbid fluid. The organs exhibit the changes commonly found in acute infections. In rapidly fatal cases the lesions may be very slight.

Symptoms.—Common Form.—The disease usually begins abruptly with a chill, followed by vomiting, excruciating pain in the head, back, and limbs, and fever. The muscles of the neck and back become rigid and contracted, so that the head is bent backward and the back is straightened; in severe cases the body may be arched in a state of opisthotonos. Kernig's sign is an almost constant phenomenon.¹ The temperature is irregular in its course and indefinite in its duration. Ordinarily it ranges between 101° and 103° F., but in some cases it remains nearly normal, and other cases it is very high. It may become normal within a fortnight or it may persist for months. The pulse is also variable. It is usually accelerated,

¹ This consists in an inability to straighten the leg completely when the patient is in the recumbent posture and the thigh is flexed at a right angle with the pelvis.

but it may be very slow (60-50). In advanced cases the respiration is sometimes of the Cheyne-Stokes type. The abdomen is often retracted and the bowels are usually constipated. The urine may be slightly albuminous. Leukocytosis (20,000 to 40,000) is the rule. Delirium is rarely absent and in severe cases it is followed by stupor and coma. Convulsions often occur. In the majority of cases emaciation is rapid and progressive.

Involvement of the Cranial and Spinal Nerves.—Evidence of involvement of the cranial nerves may be found in nystagmus, strabismus, ptosis, inequality of the pupils, and partial deafness or blindness. Involvement of the spinal nerves is shown in extreme cutaneous hyperesthesia, tonic spasms of the flexor muscles, and later, at times, in paralysis of the extremities.

The Eruption.—The eruption is neither constant nor characteristic. In many cases a blotchy purpuric eruption appears over the entire body. Herpes facialis is also frequently observed. In other cases an urticarial or a roseolar rash appears.

Lumbar Puncture.—Fluid obtained by lumbar puncture is usually turbid and contains the diplococcus and a preponderance of polynuclear cells.

Fulminant Form.—There is an abrupt onset, with a chill, followed by vomiting, headache, moderate fever, convulsions, a petechial or purpuric rash, and death within twenty-four or thirty-six hours from collapse.

Abortive Form.—The disease begins abruptly with grave symptoms, but terminates in a few days in recovery.

Intermittent Form.—In this form intermissions or marked remissions occur daily or every other day in the fever and other symptoms.

Chronic Form.—In some cases the acute symptoms subside, but the patient remains in a stuporous state for many months and ultimately presents extreme emaciation.

Diagnosis.—*Typhoid Fever.*—The gradual onset, the regular fever, the diarrhea, and tympanites, the Widal reaction, and

the absence of rigidity, of intense pain in the back and limbs, of facial palsies, of leukocytosis, of Kernig's sign, of herpes, and of changes in the spinal fluid will serve to distinguish typhoid from cerebrospinal fever.

Typhus Fever.—The regular fever, the absence of intense pain in the back and limbs, of facial palsies, of Kernig's sign, and of muscular rigidity will distinguish typhus from cerebrospinal fever.

Acute articular rheumatism may resemble cerebrospinal meningitis, but the early involvement of the joints, the acid sweats, and the absence of rigidity, of eruption, and of facial palsies, will distinguish it from cerebrospinal meningitis.

Tuberculous Meningitis.—In this disease the onset is less abrupt; there is less tendency to opisthotonos; herpes is rare; petechiæ are absent. Lumbar puncture affords a reliable means of diagnosis and a primary focus of tuberculosis can usually be detected elsewhere in the body.

Epidemic Encephalitis.—In this disease rigidity of the neck is slight or absent, ophthalmoparesis is a very early feature, Kernig's sign is wanting, and the spinal fluid is clear, is moderately rich in small lymphocytes, and is apparently sterile.

Prognosis.—The mortality has varied in different epidemics from 50 to 75 per cent. Under serum treatment it probably does not exceed 35 per cent. The cases without severe cerebral symptoms are the most favorable.

Complications and Sequelæ.—These include defective vision from inflammation of the cornea or retina or from atrophy of the optic nerve; defective hearing from inflammation of the auditory nerve or from suppurative inflammation of the internal or middle ear; pneumonia; arthritis; asphasia; peripheral palsies; imbecility; chronic hydrocephalus; and persistent headache from chronic meningitis.

Treatment.—The patient should be isolated in a quiet, darkened, and well-ventilated room. The diet should be liquid and supporting. In some cases, in order to secure the ingestion of enough nourishment, it may be necessary to resort to nutrient

enemas or forced feeding by means of a stomach-tube. Cardiac failure must be combated by stimulants, of which the best is whisky or brandy.

In sthenic cases the withdrawal of several ounces of blood by wet-cups applied along the cervical vertebræ may prove useful. Cold applied to the head and along the spine affords considerable relief. Blisters to the nape of the neck are of doubtful value, at least during the irritative stage. Morphine hypodermically is the best drug for the relief of pain, restlessness, spasms, and insomnia. In mild cases bromids may suffice.

Repeated lumbar punctures have been found useful in relieving excruciating headache, delirium, somnolence, and coma. The serum of Flexner and Jobling has given very encouraging results. From 30 to 50 c.c. are injected in the spinal canal after an equal amount of cerebrospinal fluid has been withdrawn, and it is advised that the treatment be repeated once a day for five or six days. Intravenous injections have also been recommended. Spinal puncture itself is helpful. Warm baths (105°–110° F.) twice a day for ten minutes are of value.

Tonics—iron, strychnin, and cod-liver oil—are generally indicated during convalescence. Local palsies will require massage and electricity.

MALARIAL FEVER

(Chills and Fever; Fever and Ague; Paludism)

Definition.—An infectious disease, excited by a protozoan parasite, first described by Laveran (*Plasmodium malarie*), and characterized by splenic enlargement, fever with periodic intermissions or remissions, chills, and anemia.

Etiology.—Man becomes infected through the bite of certain mosquitos, namely, those belonging to the genus *Anopheles*, which serve as hosts for the parasite. The usual source from which the mosquito derives the parasite is man. The condi-

tions predisposing to infection are those which are favorable to anophelinæ, namely, high temperature, humidity, and natural collections of water undisturbed by winds or currents. Persons living in high-lying localities are less liable to infection than those living in low lands, because the mosquito does not rise high above the ground. Malaria is more likely to be contracted at night than during the day, because most species of anopheles are nocturnal in their habits. Males being more exposed to infection are more often attacked than females.

Parasitology.—At least three distinct species of *Plasmodium* are recognized in man: *Plasmodium vivax*, causing tertian fever; *Plasmodium malariae*, causing quartan fever; and *Plasmodium falciparum*, causing estivo-autumnal fever or tropical malaria.

The infected mosquito introduces the spores (*sporozoites*) into the system of man. In the red blood-cell the hyaline spore develops into an ameboid nucleated form, using the hemoglobin of the corpuscle as food, and finally leaving only a residue of blood-pigment (melanin). In twenty-four, forty-eight, or seventy-two hours, according to the species, the mature parasite divides into a number of daughter cells, forming a rosette around a central clump of pigment. The red blood-corpuscle then disintegrates liberating the daughter cells and also a toxin, which is the cause of the malarial paroxysm. The free daughter cells (*sporozoites*) enter other red blood-cells and repeat the sporulating cycle. Although male and female elements exist among the sporozoites, segmentation in man is wholly asexual. Sporozoites are produced sexually only in the mosquito.

In the stomach of the mosquito the male and female elements unite to form a vermicule, a mobile form, which enters the stomach-wall, where it develops into a sac (zygote) containing many spores. This sac ultimately ruptures, liberating the spores (*sporozoites*), which enter the venosalivary gland of the insect, whence they are discharged into the system of man.

The tertian parasite (*P. vivax*) has fine pigment granules, is actively ameboid, and in the segmenting form (rosette) has

from fifteen to twenty spores. The red blood-cell containing it is swollen and pale. It requires forty-eight hours to complete its life cycle in the blood; hence when a single group of parasites is present paroxysms occur every other day (tertian fever). If, however, two groups coexist and sporulate on alternate days, a paroxysm occurs daily (quotidian fever).

The quartan parasite (*P. malaricæ*) contains coarser granules, is less actively ameboid than the tertian parasite, and in the segmenting form has only from six to twelve spores. One group of quartan parasites excites a chill every fourth day (quartan fever).



FIG. 14.—Various forms of hematozoa: Tertian organisms (Thayer and Hewetson); a, young hyaline form; b, hyaline form with beginning pigmentation; c, pigmented form; d, full-grown pigmented form; e, f, g, segmenting forms.

The Estivo-autumnal Parasite (*P. falciparum*).—The latter half of the life cycle of this parasite is carried out in the internal organs. The endocorpuscular form is smaller than that of either the tertian or quartan parasite, and contains less pigment. The affected corpuscle is usually shrunk and of a “brassy” appearance or completely decolorized. Segmentation occurs only in the internal organs. After the infection has lasted a week or longer, crescentic and ovoid bodies, with central clumps of coarse pigment granules, appear in the blood. These bodies are characteristic of estivo-autumnal infection and represent sexually differentiated forms, or gametocytes, which breed in the stomach of the mosquito. There appear to be at least two varieties of the estivo-autumnal parasite, one completing its life cycle in twenty-four hours, the other, in forty-eight hours.

Pathologic Effects.—The destruction of the red cells by the parasites is followed by anemia, melanemia, and pigmentation of the organs. The spleen becomes greatly enlarged from congestion. In chronic cases (malarial cachexia) it becomes hard and tough from hyperplasia of the fibrous tissue. Extreme disintegration of the blood may occasion thrombosis of small vessels and also hemoglobinuria. Colitis and acute nephritis are not rare in severe estivo-autumnal infections.

Clinical Varieties of Malaria.—The following clinical forms are recognized: (1) Intermittent malarial fever; (2) estivo-autumnal fever; (3) pernicious malarial fever; (4) blackwater fever; (5) malarial cachexia (chronic malaria); (6) latent and masked malaria.

INTERMITTENT MALARIAL FEVER

Intermittent malarial fever is excited by tertian or quartan parasites. It is characterized by paroxysms of fever occurring at definite periods, each paroxysm consisting of a cold, a hot, and a sweating stage.

Cold Stage.—This stage is characterized by lassitude, aching in the limbs, and great chilliness. The features are pinched; the lips blue; and the surface is cold and rough (cutis anserina). The rectal temperature, however, is high (105°–106° F.). Vomiting may occur. The chill may last from a few minutes to an hour or more.

Hot Stage.—The surface temperature gradually rises; the skin becomes hot; the face flushed; the eyes injected; and the pulse full and rapid. The temperature in the axilla may reach 106° or 107° F. The patient complains of severe pain in the head, back, and limbs, and of intense thirst. The urine is scanty and dark colored. This stage usually lasts from one to five hours.

Sweating Stage.—The fever gradually subsides; the pains grow less, free perspiration follows, and the urine becomes plentiful. Within an hour or two the attack is over and the patient falls into a refreshing sleep.

In addition to the recurring paroxysms, intermittent malarial fever presents symptoms common to all forms of malarial infection, namely, enlargement of the spleen, anemia, pigmentation of the leukocytes, and the presence of the parasites in the blood. There is no leukocytosis.

ESTIVO-AUTUMNAL FEVER

(Remittent Fever ; Continued Fever)

In temperate zones this type occurs chiefly in the late summer and autumn. In tropical countries, where it often assumes a most severe form, it occurs at all seasons.

The symptoms of estivo-autumnal fever are often quite irregular. The hot stage of the paroxysm often lasts twenty-four or thirty-six hours, or even longer, and the intermissions are very short. In many cases there are no actual intermissions, but simply remissions (remittent fever). The chill and the sweat may be as severe as in intermittent fever, but usually they are slight and of short duration. There is often slight jaundice (bilious remittent fever). In some cases there is mild delirium, making the condition resemble very closely typhoid fever. Prostration is always marked. The spleen is enlarged. The characteristic parasite is found in the blood.

PERNICIOUS MALARIAL FEVER

This type is excited by the estivo-autumnal parasite. It prevails in tropical and subtropical countries, and is rare in temperate regions. The symptoms vary with the localization of the parasite. If the latter accumulate in the capillaries of the brain and meninges, the attack may be manifest by delirium, aphasia, and rapidly developing coma (*comatose type*). If the localization is gastro-intestinal, there may be vomiting and purging of serous material, cramps, suppression of urine, coldness of the surface, profuse sweating, and fatal collapse (*algid type*). In other cases, in consequence of a sudden and intense hemolysis, the paroxysms are associated

with jaundice, bilious vomiting, and hemoglobinuria. Bleeding into the subcutaneous tissues and from the mucous membranes may also occur (*hemorrhagic type*).

Blackwater or Hemoglobinuric Fever.—This condition, which is now generally conceded to be a result of malaria, occurs chiefly in tropical regions where severe forms of tertian ague prevail. It is characterized by intermittent or remittent fever, jaundice, and prostrating chills, followed by pain in the back, bilious vomiting, the passage of reddish-black urine (hemoglobinuria) and rapidly developing anemia.

Malarial Cachexia.—Malarial cachexia may be a sequel of repeated attacks of intermittent or estivo-autumnal fever, or it may develop insidiously as a primary condition.

There is pronounced anemia with its attending phenomena. Pigment granules are found in some of the leukocytes and in the plasma. The parasites are at times absent from the blood. The complexion is sallow or muddy. The temperature is usually subnormal, but there may be occasional slight attacks of fever. The spleen is greatly enlarged. Weakness and emaciation are marked. Indigestion, flatulency, and constipation are common symptoms. Periodic headache, neuralgia, and hematuria are sometimes observed.

Latent and Masked Malaria.—In latent malaria the parasites can be demonstrated in the blood, but no symptoms are present. In masked malaria, the parasites are present in the blood and symptoms are present, but the latter are atypic often taking the form of headache, neuralgia, diarrhea, or dysentery.

Diagnosis of Malarial Infections.—Estivo-autumnal fever sometimes resembles *typhoid fever*. The latter may be recognized by the marked abdominal symptoms, typical rash, Widal reaction, and the absence of the malarial parasite.

Yellow Fever.—The comparatively slow pulse, early albuminuria, bloody vomit, single remission in the temperature, and the absence of splenic enlargement and of parasites in the blood will serve to distinguish yellow fever from bilious remittent fever.

Charcot's Hepatic Fever.—The history of the case, the pain and tenderness over the region of the gall-bladder, the leukocytosis, and the absence of parasites from the blood will lead to the recognition of this condition.

Leukemia.—The enormous increase of the white cells of the blood will serve to separate myelogenous leukemia from malarial cachexia.

Prognosis.—Ordinary tertian and quartan infections offer a uniformly good prognosis. Recovery usually occurs in from two to three weeks. The outlook is also favorable in relapse. Pernicious malarial fever is an extremely grave disease, the second or third paroxysm not rarely proving fatal. In malarial cachexia the prognosis should be guarded, especially if a change of climate cannot be secured.

Treatment.—Prophylactic measures include the extermination of mosquitos, the prevention of infection of mosquitos, and the prevention of infection by mosquitos (Manson). The most useful methods of suppressing mosquitos are the efficient drainage of pools and swamps and the cultivation of damp soils. Covering the surface of the water with petroleum will also free pools from larvas for from two to four weeks. To prevent the infection of mosquitos, malarial patients should be carefully screened. The chief means of preventing infection by mosquitos are avoidance of sleeping in the open air and of exposure to the evening and early morning air, adequate protection from the insects, and the use of quinin in daily doses of from 3 to 5 grains.

Quinin is the only reliable remedy for malarial fever. Methylene-blue (2 to 5 grains with half its weight of powdered nutmeg thrice daily) and Warburg's tincture possess some value, but, being distinctly less efficacious than quinin, they should be employed only when the latter is not well borne.

In ordinary *intermittent fever* the quinin should be given in doses of 10 grains three times a day until the paroxysms cease and then in doses of 10 grains once a day for several weeks. The administration of a laxative dose of calomel as a *prelimi-*

nary measure increases the efficacy of the quinin, probably by facilitating its absorption.

For adults quinin is best prescribed in capsules, cachets, or freshly made pills. For children it may be given suspended in syrup of chocolate or elixir of licorice. During convalescence iron and arsenic may be advantageously given with the quinin, as in the following formula:

℞. Ferri pyrophosphatis..... gr. xxx
 Arseni trioxidi..... gr. ss
 Quininæ sulphatis..... gr. xl
 Pulveris capsici..... gr. x.—M.

Pone in capsulas, No. xx.

SIG.—One thrice daily after meals.

In *estivo-autumnal fever* larger doses of quinin (40 to 45 grains a day) are usually required. In *pernicious malarial fever* the patient should be cinchonized as quickly as possible by injecting at once into the tissues of the thigh or buttock 15 grains of quinin-urea hydrochlorid in 10 c.c. of water or by injecting 10 grains of quinin dihydrochlorid in 200 c.c. of saline solution cautiously 2 or 3 times a day.

Symptomatic Treatment.—During the cold stage of the paroxysms the patient should be well covered with warm blankets and given hot drinks. Opium in the form of paregoric is sometimes useful in mitigating discomfort. It may be combined with a few minims of aromatic spirits of ammonia, chloroform, or Hoffmann's anodyne. In the hot stage much relief is afforded by frequently sponging the body with cool water, giving cold drinks, and administering, if the symptoms are very severe, a small dose of acetphenetidin. In the algid type of pernicious malarial fever it may be necessary to give alcohol freely, with camphor, caffein and strychnin, to tide the patient over the paroxysm.

Malarial cachexia requires tonic and hygienic treatment. Arsenic, iron, and cod-liver oil are especially valuable. Arsphenamin is worthy of trial. As in other manifestations of malaria, quinin is indicated so long as the blood shows para-

sites. According to Wood, it is much better to produce distinct cinchonism at intervals than to give the drug continuously in moderate doses. If there is constipation, mild bitter laxatives are beneficial. Change of locality is sometimes necessary to effect a cure.

SCARLET FEVER

(Scarlatina)

Definition.—An acute, contagious disease, characterized by high fever, a rapid pulse, a punctiform scarlet rash, sore throat, and a marked tendency to nephritis.

Etiology.—The specific micro-organism of scarlet fever has not been isolated. The streptococcus pyogenes is present in most of the complicating lesions. The contagion may be transmitted from the patient to another either directly or through the medium of infected objects. Milk infected from human sources is occasionally the medium of transmission. Air-borne conveyance of the disease is rare. The disease is probably contagious at all periods. The contagiousness of the post eruptive period, which is to be ascribed to sore throat, nasal or aural discharges, rather than to epidermal desquamation, sometimes lasts many weeks. The young are especially predisposed, but not equally so. One attack usually confers lasting immunity.

Pathology.—The throat is inflamed and sometimes ulcerated; the liver and spleen are engorged; and the muscles reveal granular degeneration. The lymph-nodes in all situations are enlarged. The kidneys frequently show the lesions of hemorrhagic nephritis, the glomeruli being especially involved. The rash is rarely detected after death.

Varieties.—(1) Simple; (2) anginoid; (3) malignant.

Symptoms.—The *period of incubation* is from three to seven days; the *invasion* is sudden and marked by sore throat, vomiting and a rapid rise of temperature. In infants there is sometimes a convulsion and in adults there is often a chill.

Throat Symptoms.—These consist in pain and difficulty in swallowing; fulness and tenderness beneath the jaw; and enlargement of the lymphatic glands. The tongue is at first heavily coated, and red at the tip and edges; in a few days the coating almost entirely disappears, and the papillæ become bright red and swollen. This appearance has given rise to the term “strawberry tongue.” The pillars, tonsils, uvula, and pharyngeal vault are deeply injected and may reveal a a punctiform efflorescence before the rash develops on the skin. In severe cases the tonsils may be the seat of follicular inflammation, or may be covered with false membrane.

Eruption.—A scarlet-red punctiform rash appears at the end of the first or at the beginning of the second day, on the neck and chest, and rapidly spreads to other parts of the body. Though the forehead and cheeks are often intensely flushed, the region around the mouth usually appears pale. The rash disappears on pressure, a white line remaining for a second or two when the finger-nail is drawn through it. It may be uniform or it may occur in discrete patches surrounded by healthy skin. It lasts for from five to seven days, and is followed by flaky desquamation, which is usually completed in five or six weeks.

In some cases the rash is pale and scarcely visible, and in others it is slightly papular or vesicular (scarlatina miliaris).

Febrile Symptoms.—The fever rises rapidly, reaching its maximum (104° – 105° F.) in from twenty-four to forty-eight hours, remains nearly uniform for three or four days, and then falls by lysis. The duration of the febrile period is from seven to nine days. The pulse is very rapid—out of proportion to the fever; the respirations are hurried; the appetite is lost; the bowels are constipated; and the urine is scanty, high-colored, and often albuminous. There is a well-marked leukocytosis.

Nervous Symptoms.—Restlessness, headache, insomnia, delirium, and convulsions may occur in the course of the disease.

Convulsions developing late in the disease should suggest uremia.

Anginoid Scarlet Fever.—This form is characterized by severe throat symptoms. The tonsils are much swollen and are often covered with false membrane. The fever is high and the prostration is profound. Ulceration of the throat frequently occurs. Death may result from exhaustion, aspiration-pneumonia, or hemorrhage.

Malignant Scarlet Fever.—The onset is abrupt, with a chill, vomiting, or convulsion; the fever is very high (106° – 107° F.); the pulse is rapid and feeble; delirium sets in, and is followed by coma. Death may result before the appearance of the rash, in from twenty-four to forty-eight hours, or the rash may be petechial and accompanied by hemorrhages from the mucous membranes.

Complications.—The most common is *nephritis*. This usually develops during convalescence. As mild attacks are unassociated with constitutional symptoms, the urine should be examined daily. Severe attacks are manifested by suppression of urine, general edema, and uremic phenomena. Nephritis may be the immediate cause of death in scarlet fever, or it may become chronic. Many cases, however, end in complete recovery.

Among other complications may be mentioned endocarditis (benign or malignant); myocarditis; inflammation of the serous membranes, especially of the pleura and pericardium; suppurative otitis media; inflammation of the accessory nasal sinuses; suppurative adenitis (usually cervical); arthritis; and general septicemia.

Diagnosis.—*Acute tonsillitis* may resemble scarlet fever, especially if the former is associated with an erythematous rash; but in tonsillitis there is no history of contagion; the pulse is proportionate to the fever; the rash, if present, is not punctiform; the tongue has not the strawberry appearance; and there is little tendency to nephritis.

Diphtheria.—The onset is less abrupt; there is more prostration; false membrane, containing the Klebs-Löffler bacillus, is always present; a cutaneous rash is usually absent; and the tongue does not present a strawberry appearance.

Measles.—The sore throat is less marked; catarrhal symptoms are present; the rash appears later, is papular, and forms crescentic-shaped patches; the fever shows a decided remission on the second or third day; Koplik's spots are to be seen in the mouth, and the pulse is proportionate to the fever.

Rubella.—This may be difficult to distinguish from scarlatina, but the fever is not so high nor the pulse so rapid; the postcervical glands are more swollen; the strawberry tongue is absent; there is no tendency to nephritis, and the rash is not punctiform.

Acute Exfoliative Dermatitis.—In this disease there is often a history of previous attacks, the intensity of the eruption is out of all proportion to the degree of constitutional disturbance, sore throat and strawberry tongue are absent, albuminuria is uncommon, desquamation begins very early and the nails and hair often exfoliate.

Accidental Rashes.—Certain drugs, as belladonna, quinin, and copaiba, and certain foods, as crabs and oysters, may produce a rash like that of scarlet fever, but it is not punctiform, and is not associated with high fever, sore throat, and rapid pulse.

Prognosis.—This should always be guarded. The mortality varies in different epidemics from 5 to 30 per cent.

Treatment.—The patient should be isolated for from six to eight weeks. All articles used in the sick-room should be thoroughly disinfected before being removed. To allay itching of the skin some bland ointment (cold cream or cocoa-butter) should be applied to the patient's body at least once a day until desquamation is complete.

The diet should consist of milk, junket, kumiss, ice-cream, fruit-juices and gruels. Water should be given freely to relieve thirst and to keep the secretions active.

Vomiting will call for antiemetics—cracked ice, carbonated water, bismuth subnitrate, or diluted hydrocyanic acid.

Fever.—Tepid sponging is very grateful throughout the febrile period. Fever above 103° F. should be combated with cold packs or baths (80° F.), and by cold applications to the head.

If the temperature is not very high, a mild febrifuge such as the following will be found useful:

℞. Tincturæ aconiti..... ℥ xl
 Spiritus ætheris nitrosi..... fʒ vj
 Liquor ammonii acetatis..... q. s. ad fʒ iiij.—M.
 Sig.—Dessertspoonful with water every three hours for
 a child of five years.

Nose and throat symptoms are usually best treated by gentle irrigations with normal salt solution. In older children, if tonsillitis is severe, the following application will be found efficacious:

℞. Potassii chloratis..... gr. xx
 Tincturæ ferri chloridi
 Glycerini..... āā fʒ ss
 Aquæ..... q. s. ad fʒ ij.—M.
 Sig.—Apply to the tonsils several times a day with a cotton swab.

Cardiac weakness must be combated with such drugs as alcohol, strychnin, and digitalis.

Cerebral symptoms are best controlled by the application of an ice-cap and the administration of bromids or small doses of chloral. If the nervous symptoms are due to high temperature, cold bathing will be found effective.

In *acute otitis media* nothing affords so much relief as gently syringing the auditory canal with hot water. The application of a leech behind the ear is also useful. When the tympanic membrane bulges, indicating the presence of pent-up pus, the latter should be evacuated by puncture.

Should severe *nephritis* develop, dry cupping over the loins, followed by warm fomentations, will often prove of

value. Aperients, especially salines, are indicated. Warm baths, hot packs, or vapor-baths should be used to promote diaphoresis. If the urine is scanty, unirritating diuretics, such as potassium citrate, are of service.

MEASLES

(Rubeola; Morbilli)

Definition.—An acute contagious disease, characterized by catarrh of the respiratory tract, moderate fever, and a red papular eruption, which appears on the fourth day, lasts four or five days, and is followed by bran-like desquamation.

Etiology.—Measles is highly contagious, and is transmitted chiefly by direct or indirect contact. Transmission by fomites or by third persons rarely, if ever occurs. The contagium is apparently associated with the nasal and bronchial secretion, but it has not been isolated. Measles is most commonly observed in children, but unprotected adults are very liable to be attacked. It is endemic in large cities and every few years it becomes epidemic. One attack usually confers immunity against subsequent attacks.

Pathology.—The lesions consist in catarrh of the entire respiratory tract. Gastro-intestinal catarrh is not uncommon. In fatal cases such complications as bronchopneumonia and pulmonary collapse are frequently observed.

Symptoms.—The *period of incubation* is from seven days to two weeks. The invasion is characterized by *catarrhal symptoms*—photophobia, redness of the eyes, increased lachrymation, sneezing, discharge from the nose, hoarseness, cough, and, in older children, expectoration.

The Fever.—The temperature rises rapidly to 103° or 104° F., but on the second day there is often a decided remission which continues until the fourth day, when the eruption appears; at this time it again rapidly runs up to or beyond its original height, where it remains for three or four days and then falls by rapid lysis or crisis.

The Eruption.—This appears about the third or fourth day on the face, and rapidly spreads over the entire body. It is composed of small, dark-red, velvety papules, which tend to form groups having crescentic borders. There is often much itching of the skin. In three or four days the eruption begins to fade, and a branny desquamation soon follows.

Minute bluish-white specks surrounded by red areolæ may be seen in strong daylight on the mucous membrane of the cheeks and lips one or two days before the skin eruption appears (Koplik's skin).

Malignant or Hemorrhagic Measles.—This form occurs usually in asylums, prisons, etc., where the conditions are unsanitary. It is characterized by a petechial rash, hemorrhages from the mucous membranes, and profound prostration.

Complications and Sequelæ.—Bronchopneumonia and acute gastro-intestinal catarrh are the most common complications. Among the less frequent complications or sequelæ may be mentioned membranous or ulcerative laryngitis, inflammation of the middle ear, chronic conjunctivitis, pulmonary tuberculosis, cancrum oris, and neuritis.

Diagnosis.—*Rubella.*—The preeruptive stage is very short; fever and catarrh are slight; sore throat is marked. The rash appears on the first or second day as a diffuse red blush, or as small, pale-red spots that do not form crescentic-shaped patches; desquamation is scarcely noticeable.

Scarlet Fever.—The onset is more sudden and is marked by vomiting; there is severe sore throat instead of a general catarrh; Koplik's sign is absent, but the tongue is characteristic; the rash appears on the first or second day as a diffuse punctiform erythema; the pulse rate is out of proportion to the fever, and there is much greater tendency to nephritis.

Prognosis.—Recovery is the rule in ordinary uncomplicated cases. Complications are liable to occur, however, and render the prognosis grave.

Treatment.—The child should be quarantined for at least a week after the disappearance of the rash. The sick-room should be well ventilated and moderately darkened. At least two weeks should be spent in bed.

Milk, junket, fruit-juices, broths, eggs, and gruels are suitable forms of nourishment. Water should be proffered at frequent intervals.

Daily inunctions of the body with cold cream or cocoa-butter will serve to allay itching of the skin. If the conjunctivitis is marked, the eyes should be protected with dark glasses and frequently cleansed with a solution of boric acid (15 grains to the ounce). Hot baths and hot drinks are indicated if the rash is delayed.

High temperature is best controlled by cold sponging or cold packs. Diarrhea usually yields to bismuth subnitrate (15 to 20 grains, three or four times daily) and small doses of codein. If there is severe bronchial catarrh, expectorants with sedatives, such as paregoric, are indicated. The following mixture will be found useful:

R. Potassi citratis..... ʒij
Tincturæ opii camphoratæ..... fʒij-iv
Glycerini..... fʒj
Aquæ..... q. s. ad fʒijj.—M.

Sig.—A teaspoonful every two hours for a child of three years.

During convalescence tonics—iron, strychnin, and cod-liver oil—are indicated.

RUBELLA

(Rötheln; German Measles; Epidemic Roseola)

Definition.—An acute contagious disease resembling both scarlet fever and measles, but differing from these in its short course, slight fever, and freedom from sequelæ.

Etiology.—The disease is highly contagious and transmission is effected chiefly, if not entirely, by direct contact. It is most frequently observed in children, but unprotected adults are

not exempt. It is essentially epidemic. One attack usually protects from another, but not from measles or scarlet fever.

Symptoms.—The period of incubation is from five days to three weeks. Prodromes are slight or altogether absent. The disease begins with drowsiness, slight fever, and sore throat. The eruption appears on the first or second day, and varies considerably in its character. In some cases the rash is composed of pinkish-red, scarcely elevated papules, which are more or less discrete (*rubella morbilliforme*); in others the rash is bright red and diffuse, like that of scarlet fever (*rubella scarlatiniforme*). It spreads rapidly, lasts two or three days, and is succeeded by slight desquamation. Apart from the sore throat, the catarrhal symptoms are slight. A very constant feature is marked swelling of the postcervical glands.

Prognosis and Treatment.—Complications are rare, and in the vast majority of cases the disease ends favorably within a week. The treatment is that of measles.

SMALLPOX

(Variola)

Definition.—An acute contagious disease characterized by a sudden onset, an eruption passing successively through the stages of macule, papule, vesicle, pustule, and crust, and high fever, exhibiting a distinct remission on the third or fourth day.

Etiology.—The infection is usually conveyed by direct exposure or by clothing, bedding, etc., which have been in contact with the patient. Air-borne infection is of doubtful occurrence. Unless protected by vaccination or a previous attack, nearly everyone, from the child *in utero* to the aged, is susceptible to the contagion. The colored race seems to be especially predisposed. Epidemics occur chiefly in winter.

A certain protozoön, *Cytorrhycles variolæ*, originally described by Guarnieri and present in two forms—one cyto-

plasmic and the other intranuclear—is regarded by some authorities as the specific cause of the disease. Pyogenic cocci occur in the pocks as secondary invaders.

Pathology.—The development of the variolous vesicle is the result of a peculiar degeneration of the protoplasm of the epithelial cells of the rete mucosum. There is a reticular degeneration of the cytoplasm with a more advanced degeneration of the nucleus. Genuine pocks are frequently found in the mouth, esophagus, nose, and larynx. Even the trachea and bronchi may contain them. The spleen is engorged. Cloudy swelling is present in the parenchymatous

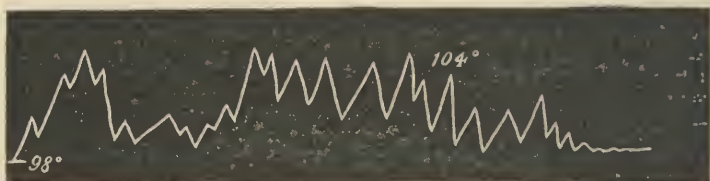


FIG. 15.—Temperature-curve in smallpox.

organs, and the spleen, bone-marrow, and the lymph-nodes are the seat of a marked cellular (mononuclear) proliferation.

Varieties.—Discrete, confluent, purpuric, varioloid.

Symptoms.—*Discrete or Ordinary Smallpox.*—The period of incubation is from ten days to two weeks. The invasion is sudden and manifested by chill, vomiting, high fever, headache, and intense lumbar pain. The temperature rises quickly to 103° or 104° F., and continues high for about three days, or until the variolous eruption is well developed, when it falls several degrees; the remission, which is accompanied by a marked improvement in the symptoms, lasts until the eruption becomes pustular (seventh or eighth day), and then the fever returns. This secondary (suppurative) fever is maintained with marked fluctuations, until the beginning of desiccation, when it gradually subsides, the temperature be-

coming normal about the end of the second week. The pulse is full and rapid (110-130); the respiration is quickened; the bowels are usually constipated; and the urine is often albuminous.

The Eruption.—In some cases a prodromal rash of a measly or scarlatiniform character is noticed on the first or second day. The true variolous eruption develops on the third or fourth day, appears first on the forehead and wrists, and spreads rapidly over the entire body. It begins as small red macules, which are quickly transformed into smooth hard papules. At the end of two days the papules become clear vesicles, centrally depressed (umbilicated), and divided by septa into several compartments, a single puncture failing to empty them. The vesicles gradually acquire a yellowish tinge and by the seventh or eighth day of the disease are converted into tense globular pustules, surrounded by red areolæ. Between the pustules the skin is edematous. In three or four days the pustules dry up or break, forming reddish-brown crusts, which emit a sickening odor and excite intense itching. In the course of two or three weeks the crusts or scabs separate leaving permanent pits or pock-marks.

Confluent Smallpox.—The papules are abundant and soon coalesce. The extremities are swollen and painful. The secondary fever is high and of long duration. True pocks nearly always develop in the air-passages and give rise to a copious fetid discharge from the nose and throat, to hoarseness, and to cough. Delirium, stupor, and subsultus are frequent symptoms. If the patient recovers, it is after a tedious convalescence, with great facial disfigurement, and often with defective vision or hearing.

Purpuric Smallpox.—In some cases the disease is ushered in with high fever, lumbar pains, and great prostration. Soon ecchymoses appear on the skin; bleeding from the mucous membranes follows; and death results before a true variolous rash appears. In other cases the disease advances like ordinary smallpox up to the pustular stage; then the pustules

becomes effused with blood, and bleeding from the mucous membranes follows. This form is also very fatal.

Varioloid.—This is modified smallpox occurring in one who has been partially protected by previous vaccination. The symptoms are mild; the eruption resembles that of ordinary smallpox, but it is usually scanty and of short duration; secondary fever is absent.

Complications.—The most common complications are boils, ulcerative laryngitis, bronchopneumonia, inflammation of the eyes (conjunctivitis, ulcerative keratitis, iritis) and otitis media.

Diagnosis.—*Varicella*.—In this disease prodromes are generally absent and the constitutional symptoms are mild. The eruption appears on the first day; it comes out in successive crops; and, unlike that of smallpox, prefers the covered surfaces. It may be maculopapular at first, but it becomes vesicular within a few hours. The lesions are superficial; vary greatly in size; are usually unilocular, and are rarely umbilicated. Desiccation begins in two or three days, the vesicles becoming irregularly puckered at the periphery and presenting a depressed blackish crust in the center—a highly characteristic appearance. In doubtful cases in children the presence of a typical vaccinal cicatrix constitutes strong presumptive evidence against variola.

Syphilis.—The history of infection; the associated evidences of syphilis (mucous patches, alopecia, etc.); the gradual onset of the illness; the slight fever; the symmetric distribution of the eruption; its dark coppery color; its polymorphous character (papules, vesicles, and pustules associated in a limited area); and the absence of itching will indicate syphilis.

Prognosis.—This depends upon the virulence of the epidemic, the vaccinal condition of the patient, and the amount of eruption. In the discrete cases the prognosis is generally favorable; in the confluent, very grave; and in the purpuric almost hopeless. In the unvaccinated the mortality ranges

between 20 and 60 per cent. Among those having one or two typical vaccine scars, the death-rate is very low—from 1 to 7 per cent.

Treatment.—The preventive measures against smallpox include the complete isolation of the patient (preferably in a special hospital), the thorough disinfection of all objects that have been in contact with him, and, above all, the vaccination of every one who has been or who is likely to be exposed to the contagion. Absolute rest in bed, light bed-clothing, a well-ventilated room of a temperature of 65° F., an easily assimilable but sustaining diet, and the free use of cool drinks are requisites of treatment. The severe lumbar pains will require opium and the application of hot-water bags. Fever is best combated by hydrotherapy—cold sponging, cold packs, or cold baths. Antipyretic drugs should be used with caution.

Gastric irritability may be controlled by diluted hydrocyanic acid (2 minims), subnitrate of bismuth (10 grains), or cocain ($\frac{1}{8}$ grain). If nervous symptoms are not relieved by hydrotherapy, opium with bromids or chloral with bromids should be tried. Alcoholic stimulants are frequently demanded, especially in confluent cases.

An attempt should be made to keep the nasopharynx clean by means of mild antiseptic sprays or douches. The eyes should also be kept clean by frequent applications of a warm boric-acid solution (15 grains to the ounce).

Prevention of Pitting.—The room should be darkened and the exposed parts covered with cloths wrung out of a weak solution of carbolic acid (1:200) or of corrosive sublimate (1:5000 or 1:10,000). Unfortunately, when the lesions are deeply seated there are no efficient means of preventing pitting.

In the stage of desiccation, warm baths followed by inunctions with cold cream or olive oil are useful in allaying itching and in hastening the removal of the crusts.

VACCINIA

(Vaccination; Cow-pox)

Definition.—A general disease with a local manifestation resembling the pock of variola, and acquired by inoculation with the virus of cow-pox. It is probably smallpox modified by passage through the cow.

History and Object.—The value of vaccination as a means of protection against smallpox was first made known to the world in a paper published by Edward Jenner in 1798.

Recent vaccination gives almost complete immunity against variola; the mortality of smallpox acquired after vaccination is almost inversely proportionate to the number of true vaccine scars.

Etiology.—Vaccinia is induced by inoculating the arm or leg with fresh virus obtained from the udder of a calf suffering from cow-pox (bovine virus). Formerly virus taken from a human vaccine vesicle was also employed (humanized virus), but on account of the risk of transmitting syphilis and other diseases, this source has been practically abandoned.

It has been shown that the addition of glycerin to vaccine lymph serves to preserve it and to free it from pathogenic bacteria.

Time of Performance.—The first vaccination should be performed, as a rule, about the fourth or fifth month, the second at the seventh year, and the third at puberty. Vaccination should always be repeated when smallpox is prevalent.

Performance of Vaccination.—The part selected should be thoroughly cleaned with soap and water, then with alcohol, and finally with pure water. A number of cross-scratches should next be made over an area about $\frac{1}{8}$ of an inch in diameter, with a sterilized needle or special scarificator, deep enough to allow of a little oozing of pinkish serum. The virus should then be applied and well rubbed into the exposed lymph-spaces by additional scarification. A shield may be worn for a

few hours or until the wound has become perfectly dry; after that it should be discarded.

Symptoms.—About the third or fourth day after the operation a papule surrounded by a red areola forms at the seat of inoculation. By the fifth or sixth day the papule becomes a vesicle with a distinct central depression (umbilication). By the eighth day the vesicle is perfected and is then surrounded by a wide zone of inflammatory edema, which is the seat of intense itching. About the tenth day the areola begins to fade, and desiccation sets in with the gradual formation of a thick brown crust, which becomes detached in the fourth week. The scar is at first red, but in the course of several months it becomes paler than the surrounding skins. It has a punched-out appearance and is pitted.

The evolution of the pock is usually accompanied by malaise, slight fever, and enlargement of the axillary glands.

Complications.—These are uncommon. Abscess, erysipelas, or even tetanus may occur from secondary infection. Roseolar or urticarial eruptions are occasionally seen. Generalized vaccina occurring spontaneously or resulting from auto-inoculation is sometimes observed.

VARICELLA

(Chicken-pox)

Definition.—An acute contagious disease of short duration, characterized by slight fever and a discrete vesicular eruption, which disappears in two or three days by desiccation.

Etiology.—The disease occurs sporadically and epidemically. It is observed chiefly in children, but adults are not exempt. One attack usually protects from others. It bears no relation to smallpox.

Symptoms.—The *period of incubation* is from fourteen to sixteen days. The constitutional disturbance is usually slight. In most cases, however, there is slight fever (99°–101° F.) with chilliness and malaise.

Eruption.—This appears within the first twenty-four hours. At first it is maculopapular, but within a few hours it becomes vesicular. The vesicles are usually sparse; are most abundant upon the trunk; come out in crops; are superficial and very variable in size; are unilocular; and are rarely umbilicated. In two or three days desiccation begins, the vesicles becoming irregularly puckered at the periphery and presenting a depressed blackish crust in the center. The crusts are thin and friable. In the majority of cases there is no marking, but occasionally a few scars or pits remain.

In rare instances gangrene occurs around the vesicles or in other parts (*varicella gangrænosa*). Nephritis, multiple arthritis, and varicellous laryngitis or conjunctivitis have occasionally been reported as complications.

Diagnosis.—The differential diagnosis between varicella and smallpox has already been considered (see p. 350).

Prognosis and Treatment.—The outlook is almost always favorable. No special treatment is required. The child should be separated from others until the crusts have disappeared. Itching may be allayed by applications of carbolized vaselin.

DIPHTHERIA

Definition.—An acute contagious disease excited by *Bacillus diphtheriæ*, and characterized by moderate fever, glandular enlargement, great prostration, anemia, and the formation of a false membrane upon certain mucous membranes, especially those of the throat and adjacent parts.

Etiology.—The disease is endemic in most large cities, and during the colder months of the year often prevails epidemically. The infection is usually transmitted by direct contact with patients suffering from diphtheria, convalescents, or healthy carriers of the infective organisms. Transmission by means of infected clothing, toys, etc., or of milk infected by carriers may also occur. Occasionally, diphtheria is acquired from infected domestic animals. Over-crowding and unsani-

tary conditions in general favor its spread. Chronic nasopharyngeal catarrh and hypertrophy of the tonsils increase the susceptibility to infection. Three-fourths of the cases are in children under ten years of age. One attack confers only temporary immunity.

The exciting cause is the Klebs-Löffler bacillus—the *Bacillus diphtheriæ*—which is found chiefly in the affected mucous membranes. The local lesions and the constitutional disturbances are caused by toxins, elaborated by the bacillus. The disease is essentially a local infection with a general toxemia. Secondary infection by pyogenic and other organisms is common.

Pathology.—The false membrane is usually found on the tonsils, pillars, and pharynx, but it may extend to the mouth, larynx, or nose. It has a grayish or yellowish appearance, is firmly attached to the underlying tissues, and when forcibly removed, leaves a raw and bleeding surface. Microscopically, it is composed of fibrin, necrotic epithelium, leukocytes and bacteria.

The lymphatic glands near the seat of infection are swollen. Focal necrosis, due to the action of the toxins, is found in the liver and other organs. The heart, kidneys, and liver are the seat of fatty and parenchymatous degeneration. Interstitial hemorrhages are frequently observed and are the result of hyaline degeneration of the capillary walls and thrombotic obstruction. Such lesions as congestion, edema, bronchopneumonia, and atelectasis are frequently encountered in the lungs.

Types.—Diphtheria may be divided according to the location of the exudate into: (1) Faucial; (2) laryngeal and (3) nasal. According to the severity of the attack it may be divided into: (1) Mild; (2) grave; (3) malignant.

Symptoms.—*Faucial Diphtheria.*—The *period of incubation* is from two to seven days. The disease usually begins with chills, moderate fever, malaise, and sore throat. The fever, as a rule, is not very high (102° – 104° F.), and its course is

quite irregular. The pulse is rapid and feeble; the bowels are constipated; the urine is scanty and frequently albuminous; and the prostration and pallor are often out of all proportion to the severity of the febrile symptoms. Moderate leukocytosis is usually observed.

Local Phenomena.—The child complains of difficult swallowing; the muscles of the neck feel stiff; there is tenderness under the jaw; the lymphatic glands are considerably swollen; and the tonsils, faucial pillars, uvula, and posterior pharyngeal wall are covered with a grayish-white membrane which, when stripped off, exposes a raw bleeding surface. The membrane may spread to the nose or larynx.

The course of the disease is indefinite, but the average duration is from ten days to two weeks.

In malignant cases there is great swelling of the inflamed areas and of the lymph nodes; the false membrane often has a gangrenous appearance and a foul odor; and the toxemia is intense. Death occurs within three or four days.

Laryngeal Diphtheria (Membranous Croup).—This is usually secondary by extension from the fauces, but it is occasionally primary. It is recognized by hoarseness or aphonia, croupy cough, progressive dyspnea, and stridulous breathing. The alæ of the nose play; the sternocleidomastoids are prominent; the suprasternal notch is deepened; and the base of the chest is retracted. Shreds of false membrane are sometimes expectorated in the violent fits of coughing. The pulse is rapid and feeble, but the temperature is rarely high. Death often results from suffocation, but recovery is not impossible even in the most unpromising cases.

Nasal Diphtheria.—This is usually secondary. When severe it is characterized by a profuse, sometimes fetid, discharge from the nose, epistaxis, and marked constitutional disturbance. The false membrane may be detected on inspection. Mild, chronic forms lasting for months, with little discharge, are sometimes met with.

Complications and Sequelæ.—The most common complications are bronchopneumonia, heart failure, the result of myocarditis or of degeneration of the cardiac nerves, acute nephritis, hemorrhage from the ulcerated surfaces, otitis media, and suppuration of the lymph nodes. The most important sequel is paralysis, due to toxic neuritis. This occurs in about 20 per cent. of the non-fatal cases and usually appears during the second or third week of convalescence. The pharynx is the most common seat, the palsy being manifested by difficulty in swallowing and the return of liquids through the nose. The external muscles of the eye are often involved the result being ptosis or strabismus. In some instances the heart is affected and if sudden death does not ensue, the condition may be manifested by tachycardia or bradycardia. The muscles of the extremities may also be involved. The paralysis usually disappears in from a few weeks to several months.

Diagnosis.—*Scarlet Fever.*—This may be distinguished by the diffuse punctiform rash, and the absence of the diphtheria bacillus.

Follicular Tonsillitis.—The differential diagnosis between this disease and diphtheria has already been considered (see p. 33).

Prognosis.—The outlook depends upon the severity of the attack and the promptness with which treatment is instituted. The disease is very fatal during the first two years of life. The average mortality at the present time ranges between 15 and 20 per cent. The nasal and laryngeal forms are always grave. Death may be due to exhaustion from the toxemia, involvement of the larynx, bronchopneumonia, cardiac paralysis, or nephritis.

Treatment.—*Prophylaxis.*—As diphtheria is prone to attack unhealthy mucous membranes, nasopharyngeal catarrh in children should receive careful attention. Infected tonsils and adenoid growths should be removed. Persons who have been directly exposed to the contagion should be passively immun-

ized by the intramuscular injection of antitoxin (500 units for infants under one year and 1000 units for older persons). By this method immunity is promptly produced, but it lasts only two or three weeks. Children in institutions and other persons who are likely to come in intimate relation with diphtheria patients, and who are found to be susceptible to infection by Schick's test* should be actively immunized by injections of a mixture of diphtheria antitoxin and toxin. Three injections ($\frac{1}{2}$ c.c. for infants and 1 c.c. for older persons) are given at weekly intervals. By this method immunity is produced in from two to three weeks and lasts, as a rule, six years or much longer.

Patients with diphtheria should be kept isolated until their throats are free from virulent bacilli. The bedroom, bedding, clothing, and all utensils used by the sick should be thoroughly disinfected.

Treatment of the Attack.—The sick-room should be well ventilated, and the temperature maintained at about 70° F. A moist atmosphere makes the breathing easier, especially in laryngeal cases. Absolute rest must be enforced. The food should be liquid or semi-solid and easily digestible. Cool water should be given freely. Antitoxin should be administered in every case at the earliest possible moment. In pharyngeal cases of moderate severity, unless seen on the first day, the initial dose should be 10,000 units. If no decided improvement follows within twelve hours, the dose should be repeated. Laryngeal cases require from 10,000 to 20,000 units, according to the time at which treatment is instituted. The injections, as a rule, should be intramuscular and preferably in the gluteal or pectoral region. In profoundly toxic cases the antitoxin may be given intravenously.

Apart from antitoxin, the most important remedies are those which tend to maintain the bodily strength. Alcoholic

* A minute quantity of diphtheria toxin is injected intracutaneously, and if the reaction is positive a red area appears in twenty-four to twenty-eight hours at the point of injection, increases in intensity during the third and fourth days, and then fades, leaving the skin brownish and scaly.

stimulants are often indicated, especially in the late stage of the disease. In septic cases alcohol is particularly well borne. Next to alcohol, strychnin $\frac{1}{120}$ grain every three or four hours) is the best stimulant. In profound adynamia, digitalis, caffein, camphor, and musk are also useful. Codein phosphate or morphin sulphate, hypodermically, is sometimes of great value in securing rest.

Local treatment is often useful in securing cleanliness of the affected parts. If, however, the applications cause violent struggling and exhaust the child, it is better to desist. Gentle irrigation of the pharynx or nose with hot saline solution (120° F.), two or three times a day, usually gives more relief than other local measure.

Externally, hot or cold applications, whichever may be the more agreeable, are useful in relieving pain and soreness in the throat. In laryngeal cases tracheotomy or intubation, preferably the latter, should not be deferred if dyspnea becomes urgent.

Convalescence must be managed with special care on account of the tendency to sudden heart-failure. Anemia will require plenty of nourishing food, and such remedies as iron, arsenic, and cod-liver oil. Paralysis usually yields to strychnin and the use of massage and electricity.

RHEUMATIC FEVER

(Acute Articular Rheumatism; Inflammatory Rheumatism)

Definition.—An acute infectious disease, characterized by polyarthritis, irregular fever, acid sweats, and a marked tendency to endocarditis.

Etiology.—The disease is most common in the second and third decades. Males are more often attacked than females. It is most prevalent in cold, damp climates and in the changeable seasons of the year.

Exposure to cold and physical depression are predisposing factors. Disposition to recurrence is a characteristic feature.

The exciting cause is still unknown. According to one view the disease is an attenuated form of septicemia, due to streptococci from diverse sources; and, according to another, it is a specific infection due to one organism—the *Diplococcus rheumaticus* of Poynton and Payne.

Pathology.—The ligaments and the synovial membrane and its fringes are congested and swollen. The synovial sac is filled with turbid fluid. Suppuration, however, never occurs and almost invariably the arthritis ends in complete resolution, although the joints may be stiff for several weeks or months. Secondary inflammations, especially endocarditis, pericarditis, and pleurisy are frequently observed in fatal cases.

Symptoms.—The disease may follow an attack of acute tonsillitis, or it may set in at once with chilliness, fever, and inflammation of the joints. The joints involved are, as a rule, the larger ones, as the knees, ankles, elbows, and wrists. They are swollen, hot, painful, and tender, but only slightly reddened. The inflammation shows a marked tendency to flit from joint to joint, and to subside in one while attacking another. In severe cases the muscles also are painful and tender. Small subcutaneous nodules are sometimes found along the tendons and over the bony prominences. The fever in ordinary cases ranges between 102° and 103° F., and is very irregular. The perspiration is often copious and has an acid reaction and a peculiar sour odor. The appetite is lost, the tongue is coated, the bowels are constipated and the urine is scanty and highly colored. Moderate leukocytosis is usually present, and as the disease progresses marked secondary anemia develops. Many *subacute cases* occur in which fewer joints are involved and the symptoms are of a mild type, but the course is often protracted.

The disease usually lasts from two to four weeks, but it may persist with alternate exacerbations and remissions for several months.

Complications.—Endocarditis, usually benign, is the most common complication, occurring in about 50 or 60 per cent. of

all cases. The lesions most frequently affect the mitral valve. Pericarditis is also common, but less so than endocarditis. Myocarditis frequently accompanies endocarditis or pericarditis. Pleurisy may also occur. Tonsillitis is not rarely present at the onset of the disease.

In children chorea may precede, accompany, or follow rheumatic fever. Hyperpyrexia (108° – 110° F.) is occasionally observed. Very rarely in adults the high fever is accompanied by delirium, convulsions and coma and to this group of symptoms the term “cerebral rheumatism” has been applied. Certain cutaneous affections, especially purpura and various forms of erythema, are sometimes observed.

Diagnosis.—*Septic Arthritis.*—This may be recognized by its association with some other septic process and by the special tendency of the inflammation to end in suppuration, which is an extremely rare termination of rheumatic arthritis.

Gonorrheal rheumatism may be recognized by the history of gonorrheal infection; by the tendency of the inflammation to spread along the sheaths of the tendons; to remain stationary in joints primarily affected, and to attack joints rarely involved in rheumatism (intervertebral, sternoclavicular, temporomaxillary); by the stubborn character of the disease, and its resistance to salicylates.

Acute Arthritis Deformans.—In this disease certain joints are likely to be affected that usually escape in rheumatism, namely, the small joints of the hands, the intervertebral, sternoclavicular, and temporomaxillary; the inflammation rarely shifts from joint to joint; the temperature is usually between 99° and 101° F., but the pulse-rate is generally elevated out of proportion to the fever; there is marked tendency to muscular atrophy, especially of the interossei muscles, and to permanent deformity and fixation of the joints; and salicylates have little, if any, effect.

Gout.—This occurs later in life, shows a decided predilection for the metatarso-phalangeal and tarsal joints, causes redness of the overlying skin, lacks high fever, acid sweats, and the

tendency to acute heart complications and is accompanied by an excess of uric acid in the blood and often by uratic deposits in the helix of the ear or elsewhere.

Prognosis.—In the great majority of cases rheumatic fever terminates in recovery. A very small number of patients die of exhaustion or some complication, such as endocarditis or hyperpyrexia with grave nervous symptoms. The disease is very prone to relapse and to recur.

Treatment.—Absolute rest in a comfortable bed is essential, and, with the view of preventing permanent injury to the heart, this should be maintained for at least ten days or two weeks after the temperature has become normal and all the arthritic symptoms have subsided. The patient should wear a loose flannel night-dress and lie between blankets. Milk, eggs, and cereals are the most suitable articles of diet. The free use of water and of lemonade should be encouraged. Salicylates have considerable power in controlling the symptoms. As a rule, about 20 grains of sodium salicylate should be given every three hours until a decided impression is made upon the disease or the phenomena of salicylism are produced, when the interval between the doses should be lengthened to four or six hours. Even in children from eight to ten years of age doses of 10 or even 15 grains every three hours are advisable. If the drug is not tolerated by the mouth 1 or 1½ drams of sodium salicylate in 150 c.c. of thin starch water may be injected into the bowel once or twice in twenty-four hours, after a cleansing soap-suds enema. Salicylate treatment should be continued for at least a week after the disappearance of the symptoms.

Alkalies, such as potassium bicarbonate or potassium citrate, in doses equal to those of the salicylate, are useful adjuvants.

R. Potassii bicarbonatis

Sodii salicylatis āā 3iv

Aquæ menthæ piperitæ f3vj.—M.

SIG.—A tablespoonful in water every three hours.

Opium, in the form of Dover's powder or of morphin hypodermically, is sometimes of great value in allaying pain, subduing restlessness, and procuring sleep. Antipyrin or acetphenetidin, in moderate doses, is also useful. When adynamia is marked, quinin (5 grains) is frequently beneficial. Anemic patients are benefited by iron.

Hyperpyrexia is best controlled by the cold bath. Endocarditis and pericarditis rarely require special remedies. The importance of prolonged rest in cases in which the heart becomes affected cannot be overestimated. During convalescence tonics, as iron, quinin, and arsenic, and a liberal diet are necessary.

Local Treatment.—In mild cases the joints may be painted with iodine and wrapped in cotton-wool. In severe cases small blisters are of great utility.

Cloths kept moistened with methyl salicylate, saturated solution of magnesium sulphate, or lead-water and laudanum (ice-cold or hot), or ointments of salicylic acid are often serviceable.

R.	Acidi salicylici.....	℥iss
	Olei terebinthinæ.....	f℥j
	Adipis benzoinati.....	q. s. ℥ij.

Sig.—Spread on lint and keep in place by means of a flannel binder.

No matter what local remedy is selected, it is highly important that the affected joints should be kept at complete rest. This may be accomplished by means of padded splints and a roller bandage.

Lingering swelling will often yield to an ointment of mercury and belladonna, with firm strapping of the articulation. Blisters are also useful. If the effusion is very great and persistent, it may be necessary to aspirate the joint.

For the stiffness of the joints massage, warm baths, and inunctions with the ointment of iodine will be found useful. The hot-air treatment also does good in some cases.

ERYSIPELAS

Definition.—An acute infectious disease excited by the *Streptococcus pyogenes*, and characterized by high fever and a peculiar inflammation of the skin and subcutaneous tissues.

Etiology.—The disease is somewhat contagious, and the poison can be carried in fomites. Certain families and certain individuals seem particularly predisposed. Puerperal women and wounded persons are very susceptible. Diseases which lower the vitality, especially nephritis, predispose. One attack does not protect against a recurrence. Erysipelas was formerly divided into traumatic and idiopathic varieties; but the two are identical, and it is probable that in those cases in which there is no conspicuous wound there is a slight abrasion through which the infection gains admittance. The exciting cause of the disease is the *Streptococcus pyogenes*.

Pathology.—Erysipelas most frequently manifests itself on the face. The affected area is bright red in color, swollen, indurated, and sharply circumscribed. The various strata of the skin are infiltrated with serum, and leukocytes and streptococci are found in the lymph-spaces. In severe cases the inflammatory products are converted into pus, and abscesses form.

Symptoms.—The *period of incubation* is from three to seven days. Prodromes are sometimes present, and consist of slight fever, chilliness, malaise, and tingling in the part that is to be affected. In many cases the disease is ushered in suddenly with a chill, followed by pain in the head and limbs, and a high, irregular fever. The temperature may reach 104° or 105° F. in twenty-four hours. The pulse is full and rapid; the tongue is heavily coated; the appetite is lost; the bowels are constipated, and the urine is scanty and often slightly albuminous. In severe cases a typhoid condition, manifested by delirium, subsultus tendinum, a dry, brown tongue, etc., not rarely develops. There is usually a marked leukocytosis.

Local Phenomena.—The inflammation usually begins in the neighborhood of the nose, and spreads upward and laterally over the head to the neck, where it frequently stops. The affected part has a crimson hue; it is swollen and tense, and frequently ends in a sharply defined ridge, beyond which, however, projections can be felt advancing into the subcutaneous tissue. The surface of the inflamed patch is at first smooth and glazed, but later it is covered with minute vesicles or blebs. The patient complains of burning and tingling; the surrounding parts are extremely edematous, so that the features may be scarcely recognizable. In four or five days the redness begins to fade and the swelling to subside; desquamation follows; the general symptoms improve; and the fever falls by crisis or rapid lysis. The average duration is from ten days to two weeks. In the so-called migratory cases, in which there is a successive involvement of new areas of integument, the disease may continue for several weeks. Relapses are very common.

Complications.—These are not common. Septicemia, ulcerative endocarditis, nephritis, arthritis, edema of the larynx, pneumonia, and meningitis are occasionally seen.

Diagnosis.—*Erythema.*—The absence of high fever, of marked swelling, and of an abrupt ridge will serve to distinguish erythema from erysipelas.

Acute Eczema.—The swelling is less marked; the itching is intense; the swelling and redness are not circumscribed, but shade gradually into healthy tissue; and there is no fever.

Prognosis.—In the robust the prognosis is favorable. In the aged, in alcoholic subjects, and in those suffering from chronic nephritis the prognosis must be guarded. Migratory erysipelas may kill by exhaustion. The average mortality is about 6 per cent.

Treatment.—As in other contagious diseases, isolation and disinfection are the most important prophylactic measures. Especially necessary is it to guard parturient and surgical patients from the contagion.

A supporting liquid diet should be given. Alcoholic stimulants are sometimes required. High fever is best controlled by cold sponging or the cold pack. Restlessness, delirium, and insomnia will call for applications of ice to the head, and perhaps the administration of morphin, chloral, or bromids.

Of the numerous special remedies recommended for erysipelas, the one which has enjoyed the most favor is the tincture of ferric chlorid (15 to 30 minims every three hours).

Local Treatment.—Among the numerous local applications recommended may be mentioned: Lotions of lead-water and laudanum, of carbolic acid (1:40), of picric acid (1:100), of magnesium sulphate (saturated solution), and of sodium salicylate (1:20). In the hands of the author ointments of ichthyol (20 per cent.) and of soluble silver (unguentum Credé) have proved most satisfactory.

The following combination often acts well:

R.	Ichthyol.....	gr. xxx
	Resorcinolis.....	ʒss
	Unguenti hydrargyri.....	ʒiv
	Adipis lanæ hydroxi.....	ʒv.—M.
		(ROSWELL PARK.)

Local abscesses should be incised and treated antiseptically. Extension to the nose and throat will call for antiseptic sprays or washes.

PYOGENIC INFECTIONS

(Septicemia; Pyemia; Septicopyemia)

Definition.—Local or general non-specific infections caused by the entrance and growth within the body of the different pyogenic micro-organisms—most frequently the *Streptococcus pyogenes* or the *Staphylococcus pyogenes aureus*, but in some cases the *Diplococcus pneumoniae*, *B. coli communis*, *B. pyocyaneus*, or *Micrococcus gonorrhææ*. Mixed infections are common.

Etiology.—The infection may be acquired by inhalation or ingestion, but in the great majority of cases it is acquired by inoculation through a wound or an abraded surface. Susceptibility to infection is much enhanced by physical debility from any cause, although robust persons are by no means immune. In many cases pyogenic infection is a secondary or terminal event in the course of some other disease, such as chronic nephritis, arteriosclerosis, diabetes, etc. As a rule, the general infection is preceded by a local lesion, unobtrusive although it may be, as a furuncle, an infected Fallopian tube, a small tuberculous cavity, otitis media, cholecystitis, etc. Occasionally no portal of entry is discoverable, and to this group of cases the term *cryptogenetic septicemia* has been applied.

Pathology.—A general intoxication resulting from the absorption of soluble bacterial products formed only at the point of invasion is referred to as a *toxemia*. The group of symptoms associated with a general invasion of the blood and tissues by pyogenic organisms and therefore due to toxins elaborated in all parts of the body is described by the term *septicemia* (bacteriemia). If in the course of such a general invasion of the blood and tissues by pyogenic bacteria multiple secondary foci of suppuration occur, the condition becomes a *pyemia* (septicopyemia). These secondary foci include abscesses, ulcerative endocarditis, suppurative pylephlebitis, etc.

The lesion of a septicemia cannot be distinguished from those of a toxemia, but vary in each condition according to the character of the infecting bacterium. The spleen is usually more or less swollen, softened, and congested. The liver, heart and kidneys present the appearances due to cloudy swelling and fatty degeneration. The liver frequently shows in addition small areas of necrosis. The lungs are congested and often present areas of pneumonic consolidation. Minute hemorrhages are sometimes observed beneath the skin and the serous membranes and within the substance of the various organs. In pyemia the lesions are similar, but in addition,

secondary foci of suppuration, usually of embolic origin, are found in the lungs or in the territory of the general circulation.

Symptoms.—The symptoms vary with the severity and character of the infection, and those of toxemia differ from those of septicemia only in intensity. In mild cases there are chilliness, fever, reaching 101° or 102° F., headache, muscular pains, anorexia, a hot, dry skin, and restlessness. These symptoms usually subside rapidly after local treatment. In severe attacks the onset is marked by a sharp chill and a sudden rise of temperature to 104° F. or higher. The chills frequently occur at intervals and the temperature continues high with daily remissions or even intermissions. The usual concomitants of fever are present and vomiting and diarrhea frequently occur. The mind may remain clear, but in many cases there is early delirium. The skin may be slightly jaundiced or show petechiæ, polynuclear leukocytosis is present, and pronounced anemia soon develops. As the intoxication progresses a state of collapse may supervene and terminate fatally within a few days or a typhoidal condition may develop and continue for several weeks. Occasionally, as in certain cases of malignant endocarditis (see p. 214), the course is chronic and for a long time marked only by a daily rise of temperature, an occasional chill, and increasing pallor and weakness.

Pyemia presents all the phenomena of severe septicemia and in addition the symptoms arising from secondary foci of suppuration in the lungs, kidneys, liver, spleen, glands, etc. Erythematous rashes are not infrequent. Death usually occurs in from two to three weeks.

Diagnosis.—This is based upon the discovery of a primary focus of infection and the general symptoms—chill, irregular fever, sweats, leukocytosis, pallor, weakness and emaciation. Malaria, typhoid fever, and acute miliary tuberculosis are the diseases most likely to come into question. The differentiation between a toxemia and a septicemia is impossible without

the aid of blood cultures. The diagnosis of pyemia is made certain by the discovery of secondary suppurative foci.

Prognosis.—This depends upon the type and severity of the infection, the resistance of the individual and the possibility of removing the infectious foci. True septicemia (bacteriemia), especially if due to the streptococcus, is always very grave. Pyemia accompanied by ulcerative endocarditis, meningitis, suppurative pyelophlebitis, or multiple visceral abscesses is invariably fatal.

Treatment.—The chief indications are to evacuate and drain all suppurative foci that are accessible and to support the strength of the patient by good hygienic conditions, careful nursing, nutritious, easily assimilated food, and such remedies as alcohol, tincture of ferric chloride, quinin, and strychnin. The results of serum treatment have not been very encouraging. Vaccines, especially those derived from a focus of infection in the individual under treatment (autogenous vaccines), are sometimes helpful; rarely so, however, except in cases which show a tendency to chronicity.

INFLUENZA

(La Grippe; Catarrhal Fever; Epidemic Catarrh)

Definition.—An acute infectious and contagious disease, characterized by fever, marked prostration, severe muscular pains, and catarrhal inflammation of the respiratory and gastro-intestinal tracts.

Etiology.—Influenza occurs endemically, epidemically, and often pandemically. The infection spreads with great rapidity, and may affect a large proportion of the population almost simultaneously. Outbreaks occur most frequently in colder weather and usually last about six or eight weeks. The disease is transmitted by contact and also by fomites. Doubtless "carriers" play an important part in disseminating it. No age is exempt, and one attack instead of conferring immunity predisposes to others. The exciting cause of influ-

enza is generally believed to be the minute bacillus described by Pfeiffer in 1892, and which is present in the nasal secretion, sputum, pneumonic exudate, and, occasionally, the blood. However, secondary invasion by other organisms, especially pneumococci, streptococci, and staphylococci, is frequently responsible for the serious effects of the disease observed in some epidemics, and influenza-like outbreaks undoubtedly occur in which Pfeiffer's bacillus plays no part.

Pathology.—Influenza does not often kill except by its complications. The latter are most frequently associated with the respiratory tract, and consist chiefly of pneumonia and its sequels, abscess of the lung, and pleurisy.

Symptoms.—The period of incubation is usually from two to four days. The disease begins abruptly with lassitude, chilliness, severe pain in the head and back, fever ranging between 102° and 104° F., and marked prostration. The catarrhal symptoms comprise injection of the eyes, sneezing, hoarseness, and harsh paroxysmal cough. In ordinary cases the temperature falls in three or four days by rapid lysis, but complications may prolong the attack for several weeks. As a rule, leukocytosis is absent. Relapses are common.

In some cases the catarrh of the respiratory tract is the chief feature; in others the gastro-intestinal tract is attacked, and the symptoms resemble cholera morbus; in a third group neuralgic pains in the head, back, and limbs are the most striking phenomena. A chronic pulmonary form has been described which is indistinguishable from tuberculosis except by bacteriologic tests.

Complications.—The most common are: lobar pneumonia, bronchopneumonia, otitis media, conjunctivitis, neuritis, and functional disturbances of the heart. Occasionally endocarditis, myocarditis, thrombophlebitis, meningitis, or temporary insanity occurs.

Diagnosis.—*Acute Bronchitis.*—The fever is not so high; there is little or no prostration; and the pains in the head and back are not nearly so marked as in influenza.

Typhoid Fever.—The gradual onset, typical temperature-curve, epistaxis, Widāl reaction, and rash will indicate typhoid fever.

Prognosis.—The prognosis is usually good, except in the aged and infirm in whom pneumonia and other complications are common.

Treatment.—Isolation, absolute rest in bed even in the mildest cases, sterilization of the secretions, regulation of the diet and careful nursing are the important general measures.

In mild cases a hot foot bath, some mild refrigerant, such as a spirit of nitrous ether or solution of ammonium acetate, and at night a dose of Dover's powder (5 to 10 grains) will usually suffice. If there be constipation, a few fractional doses of calomel may be given with advantage.

In more severe cases quinin (2 to 5 grains thrice daily) may be given throughout the attack. Pains are controlled to some extent by acetphenetidin with salicylates or benzoates.

The following combination is often useful:

℞. Acetphenetidini
Salophen
Sodii benzoatis. āā ʒj.—M.

Fiant chartulæ No. xii.

SIG.—One every three or four hours.

If the suffering is intense, morphin should be used hypodermically. Violent headache is treated best by small doses of phenacetin and the application of an ice-cap to the head. Heart-failure should be combated by alcohol, ammonia, and strychnin. Bronchial catarrh will require the remedies indicated in simple bronchitis.

MUMPS

(Epidemic Parotitis)

Definition.—An acute contagious disease, characterized by inflammation of the parotid and other salivary glands.

Etiology.—The disease occurs sporadically and epidemically. It is most frequently observed in young children, but unpro-

tected adults are not exempt. Males are more susceptible than females. The disease is highly contagious, and the virus is probably contained in the saliva, but it has not been isolated. One attack usually confers immunity.

Pathology.—The lesions consist of congestion and edema of the parotid gland and periglandular tissues, and catarrhal inflammation of Stensen's duct. The process resolves without suppuration.

Symptoms.—The *period of incubation* is from ten days to three weeks. The disease is ushered in with chilliness, malaise, and moderate fever (101° – 103° F.), followed by swelling of the parotid gland. The swelling is observed below and in front of the ear, is pyriform in shape, and has a doughy feel. The surrounding tissues are edematous, the submaxillary glands are often swollen, and the features may be distorted beyond recognition. The movements of the jaw are restricted and painful. The salivary secretion may be either increased or decreased. After an interval of a day or two the opposite gland usually becomes affected. The disease lasts a week or ten days and almost always ends in recovery.

Complications.—Orchitis, unilateral or bilateral, is common after puberty. It usually follows the parotitis but it may accompany it or even precede it. Atrophy of the testicle sometimes supervenes. In girls a transference of the inflammation to the vulva, mammary gland, or ovary is occasionally observed. Rare complications comprise maniacal delirium, otitis media, meningitis, nephritis, optic neuritis, pancreatitis (mild form), and chronic hypertrophy of the glands.

Treatment.—The patient should be kept in bed. Isolations should last three weeks from the onset of the disease. Mild aperients and refrigerants are useful. If the pain is severe, hot fomentations or a saturated solution of Epsom salt on several thicknesses of gauze may be applied. In mild cases, covering the gland with cotton batting will suffice.

Orchitis will require rest, suspension of the affected gland, and the application of a saturated solution of Epsom salt or,

better still, of an ointment of guaiacol (10 to 15 per cent.). After the tenderness has subsided, an ointment of mercury and belladonna will be found useful in reducing the swelling.

YELLOW FEVER

Definition.—An acute infectious, endemic or epidemic disease, characterized by fever of one or two paroxysms, jaundice, albuminuria, and a marked tendency to hemorrhage, especially from the stomach.

Etiology.—The specific organism of yellow fever is not definitely known, but it is probably a spirochete—*Leptospira icteroides*. Man is inoculated through the bites of a certain species of mosquito—*Stegomyia calopus*—which serves as the intermediate host for the parasite. The mosquito is infected only by biting a yellow-fever patient during the first three days of the disease and cannot transmit the infection until a period of from twelve to twenty days has elapsed. The disease is not conveyed by fomites.

Yellow fever occurs endemically in certain tropical sea-ports (West Coast of Africa, maritime towns of Central America), whence it occasionally spreads to temperate zones. The predisposing factors are those which are favorable to the growth of mosquitos—high temperature, surface drainage, and swampy soil. The colored race is less susceptible than the white. One attack usually confers immunity.

Pathology.—The tissues are stained yellow. The liver presents a mottled reddish-yellow (autumn-leaf) hue, and is the seat of extensive fatty degeneration. The kidneys usually show the lesions of acute hemorrhagic nephritis. The gastrointestinal mucous membrane is swollen, congested, and frequently infiltrated with blood. The heart-muscle is pale and fatty.

Symptoms.—The *period of incubation* is from two to five days. The disease begins with a chill, followed by pain in the head, back, and limbs. The temperature rises rapidly until it reaches its maximum (103°–105° F.). The pulse is at first

accelerated, but as the temperature rises it shows a marked tendency to fall, sometimes dropping in grave cases to 60 or even 50 a minute by the third day (Faget's law). The face is flushed; the conjunctivæ are injected; the pupils are small; the tongue is coated; the epigastrium is tender; the stomach is irritable and unretentive; the bowels are constipated; and the urine is scanty and often albuminous by the end of the first day.

Jaundice is rarely marked before the second or third day, although a slight icteroid tinge of the conjunctivæ is often noticeable within the first twenty-four hours. The first stage usually lasts from two to three days, and is followed by a rapid fall in the temperature and an improvement in all the symptoms (stage of calm or remission). At this time convalescence may begin or the patient may pass into the second stage.

The *second stage* is characterized by deep jaundice, persistent vomiting, vomiting of dark blood (*black vomit*), marked albuminuria, and often by suppression of urine and hemorrhages from the mucous surfaces. The mind usually remains clear until very near the close, but in some cases delirium and stupor develop. This stage may be afebrile, and not infrequently the temperature rises again after the period of calm, while the pulse remains extremely low (50 to 40 a minute). Death usually results from collapse or uremia. The duration of the disease is from three to ten days.

Diagnosis.—*Dengue.*—This disease does not exhibit a slow pulse with a rising temperature, early albuminuria, jaundice or black vomit.

Acute Yellow Atrophy of the Liver.—The rapid pulse, the diminution in the size of the liver, the slight fever, the marked cerebral symptoms, and the presence of leucin and tyrosin in the urine will indicate acute yellow atrophy.

Malaria.—This may be distinguished by the enlargement of the spleen, the multiple remissions, the presence in the blood of the parasites, and by the absence of black vomit.

Prognosis.—This should always be guarded. The average mortality in different epidemics is from 20 to 70 per cent. In individual cases high fever, a very slow pulse, marked cerebral symptoms, black vomit, and suppression of urine are unfavorable features.

Treatment.—"The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitos and the protection of the sick from the bites of these insects."¹

Absolute rest in a quiet, well-ventilated room and careful nursing are essential. Only the blandest food should be allowed. Many clinicians of wide experience advocate the withholding of all food during the first day or two. For the gastric irritability a sinapism may be applied to the epigastrium, and cracked ice, champagne, hydrocyanic acid, or cocain may be given internally. Fever is best controlled by the external application of cold. Suppression of urine will call for dry cupping over the loins, alkaline diuretics, hot-air baths, and subcutaneous or rectal injections of warm saline solutions. Remedies have little effect upon the black vomit. Tincture of ferric chlorid, adrenalin solution (1:1000), and oil of turpentine have been recommended.

PULMONARY TUBERCULOSIS

Definition.—A specific inflammatory disease of the lungs caused by the *Bacillus tuberculosis*.

Etiology.—The disease most commonly develops between the ages of fifteen and forty. Though tuberculosis is very rarely transmitted from parent to offspring, an inherited susceptibility to the disease is not rarely observed. Overcrowding, bad ventilation, lack of sunlight, and poor food; occupations that necessitate the breathing of impure air and irritating dusts; a small, flat chest; and certain other diseases, such as catarrh of the respiratory tract, whooping-cough, measles, diabetes, and cirrhosis of the liver, favor infection.

¹Report of the U. S. Army Commission, *Jour. Hyg.*, vol. ii.

Infection may take place: (1) By the inhalation of air laden with moist particles of infected sputum, expelled in coughing, sneezing, etc. (droplet infection), or with the dust of dried tuberculous sputum; (2) by the ingestion of food contaminated directly or indirectly with infected sputum, as the milk, or rarely the meat, of tuberculous cattle, or of bacilli-infected material that has been conveyed to the mouth by the fingers, drinking-cups, toys, etc.; (3) by the direct inoculation of wounds (rare); (4) by direct parental (placental) transmission (very rare).

The bacilli may reach the lungs directly through the air-passages, or they may be brought to these organs from the intestines, tonsil, or some other portal of entry by the lymphatics or blood-vessels.

Pathology.—The exciting cause is the *Bacillus tuberculosis*, a minute rod, about one-fourth or one-half as long as the diameter of a red blood-cell, and often slightly bent and beaded. Its detection depends on the power of the stained bacillus to resist the decolorizing effects of acids. Several types of bacilli exist, but only two, the human and the bovine, appear to be infectious for human beings. The bovine organism, which is distributed chiefly by means of milk, is not an uncommon cause of tuberculosis in young children, but only a very small proportion of cases in adults are due to this type.

The lodgment of the bacilli in the terminal bronchioles or peribronchial tissues excites a proliferation of the fixed connective-tissue cells. The new cells, from their resemblance to epithelial cells, are known as *epithelioid cells*. They have a relatively large amount of protoplasm and a rather faintly staining vesicular nucleus. *Giant-cells* are sometimes formed by the fusion or overgrowth of the epithelioid cells. In consequence of the local irritation the focus of epithelioid cells is soon surrounded by a wall of *small round cells* (lymphocytes and proliferated fixed connective-tissue cells), the whole forming a gray, translucent mass—the gray tubercle of Laënnec. In short time the tubercle toxin brings about necro-

tic changes in the cellular accumulation, and these gradually transform the tubercle into a yellow, cheesy mass—the yellow tubercle of Laënnec. The degenerated tubercles fuse and form the cheesy masses so commonly observed at the autopsy. At this stage one of two things may occur: the mass may soften, discharge through a bronchial tube, and leave behind a cavity with ulcerating walls, or it may become encapsulated by an overgrowth of connective tissue and subsequently calcified.

The destructive changes are usually due to the association of pyogenic cocci with the tubercle bacilli. In some very acute cases of tuberculosis the infiltration is not in the form of definite tubercles, but is diffuse, the entire mass at first presenting a peculiar grayish-red gelatinous appearance, and later becoming distinctly caseous.

In addition to the specific lesions other secondary changes are noted. The lung tissue adjacent to the tuberculous deposits is often the seat of a true pneumonic inflammation; the connective tissue is almost always more or less increased; the bronchi are inflamed; and the pleural surfaces over the affected areas are frequently adherent.

Forms.—Two chief forms of pulmonary tuberculosis are recognized: The acute and the chronic. Acute tuberculosis occurs in three subtypes. In the *acute pneumonic* type a large area of the lung is uniformly infiltrated as in croupous pneumonia, and the process rapidly proceeds to caseation and softening. In the *bronchopneumonic* type the infiltration occurs in more or less discrete patches surrounding the final distributions of the bronchi. By fusion of the foci a lobe or even an entire lung may ultimately become involved. In the *acute miliary* type the lungs are studded with countless discrete tubercles of the size of a millet seed or a little larger. This condition which is often secondary to chronic tuberculosis of the lung, is usually a part of a widespread infection involving many organs (general miliary tuberculosis), dissemination of the bacilli having taken place through the blood stream.

Chronic ulcerative tuberculosis, the most common form of pulmonary tuberculosis, begins with the development of gray tubercles, usually in the apical region, and tends to spread downward, various forms of lesions—cavity, caseation, conglomerate tubercles, discrete fresh tubercles—often being found at autopsy in the same lung. In many cases, however, the infection is arrested at any early stage, and in this event the only lesion at death may be a small cheesy or calcareous mass surrounded or pervaded by fibrous connective tissue. In some instances the term *chronic fibroid tuberculosis* is applicable, as the progress of the disease is held in abeyance for years by an enormous hyperplasia of connective tissue surrounding and isolating the tuberculous areas. Bronchiectasis and pleuritic adhesions are always present.

Symptoms of Acute Pneumonic Tuberculosis.—Clinically this form in the beginning closely resembles lobar pneumonia or bronchopneumonia, but the symptoms instead of subsiding at the end of ten days or two weeks persist and gradually become more or less characteristic. The fever assumes a remittent or an intermittent type, chills and sweats occur, signs of softening and excavation develop in the affected lung, anemia and emaciation become pronounced, and death results in from four to eight weeks; or, more rarely, a certain degree of improvement occurs and the clinical picture slowly changes to that of chronic ulcerative tuberculosis.

Acute Miliary Tuberculosis.—The symptoms of acute miliary tuberculosis are described on page 386.

Symptoms of Chronic Ulcerative Tuberculosis.—The onset is usually insidious and marked by pallor, gastric disturbance, loss of flesh and strength, and by a dry, hacking cough that is noted especially in the morning. From some undue exposure the cough is often aggravated, and to this obstinate “cold” the disease is usually attributed. In some cases the symptoms appear abruptly with hemorrhage or an acute pleurisy. Occasionally the first manifestation is gradually developing hoarseness.

Slight fever and acceleration of the pulse are early symptoms of great diagnostic import. The temperature is marked by an evening exacerbation, during which the face is flushed, the eyes are bright, and the mind animated. As the disease advances the cough becomes troublesome and the expectoration more abundant. In developed cases the expectoration is greenish-gray in color, is in coin-shaped plugs (nummular), is heavy and sinks in water, is often blood-streaked, and on microscopic examination is found to contain bacilli and fibers of elastic tissue.

Pleuritic pains are often present. Hemoptysis occurs in from 50 to 60 per cent. of all cases. In the later stages the bleeding is sometimes profuse, although it is not often immediately fatal. Vomiting, the result of the cough or of secondary gastric disease, is common. The respiration is accelerated, but patients rarely complain of dyspnea except upon exertion.

The final stage is characterized by extreme emaciation, weakness, pallor, remittent or intermittent fever of a hectic or septic type, and sometimes edema of the feet. The mind is usually clear and peculiarly hopeful to the end.

PHYSICAL SIGNS.—The chest may be well formed. Often, however, it is long and flat, with hollow supraclavicular and infraclavicular spaces, prominent scapulæ, and oblique ribs. When the disease is well advanced, there may be retraction with diminished expansion over one apex. Slender tapering fingers with incurving of the nails, or, on the other hand, short broad finger-tips ("clubbed" fingers) are often noted.

Palpation.—This reveals imperfect expansion and exaggerated vocal fremitus.

Percussion.—Dulness over the solidified areas can be detected at an early period of the disease. It may be obtained above or below the clavicles, in the supraspinous fossæ, between the scapulæ, or in front, near the sternal border.

Cavities unless they are filled with fluid, yield a tympanitic sound, or if thin-walled, a so-called cracked-pot note. The

latter is best obtained with quick, light percussion strokes, when the patient's mouth is open.

Auscultation.—In the earliest stage the respiratory sounds may be feeble over the affected area. Later the breathing is harsh and the expiration is prolonged (bronchial). The vocal resonance is increased. Crackling râles are usually audible, especially after coughing. Auscultation over cavities may reveal cavernous or amphoric breathing, bronchophony or pectoriloquy, and large gurgling râles.

Anomalous Physical Signs.—The vocal fremitus is diminished when there is much pleural thickening. Normal resonance or hyperresonance may replace dullness when there is much emphysema between small tuberculous foci. Weak breathing may replace bronchial or cavernous when the tubes or cavity are filled with mucus. The signs of cavity are sometimes produced by consolidation in the neighborhood of a large bronchus.

Chronic Fibroid Tuberculosis.—This form may last many years. It is characterized by gradual loss of flesh and strength, dyspnea, and mucopurulent expectoration which is at times fetid from retention in bronchiectatic cavities. Fever and sweating are usually slight. Hemoptysis is somewhat frequent. Clubbing of the fingers is often marked. The physical signs are much like those of chronic ulcerative tuberculosis, but retraction and deformity of the chest-wall is a more conspicuous feature.

Complications.—The chief complications of pulmonary tuberculosis are hemoptysis, bronchopneumonia, pleurisy, pneumothorax or pneumopyothorax, gastro-intestinal catarrh, rectal fistula, amyloid degeneration of the viscera, tuberculosis of other structures (larynx, meninges, intestines, kidneys, peritoneum, etc.), and general miliary tuberculosis.

Diagnosis.—Slight fever, especially toward evening, a frequent irritable pulse, persistent cough, progressive loss of weight, hemoptysis, signs of infiltration of the lung and the occurrence of so-called idiopathic pleurisy are suggestive

features. The various tuberculin tests may be employed in doubtful cases, but their value should not be overestimated as they are not uncommonly positive in apparently normal individuals. The presence of tubercle bacilli or of elastic fibers in the sputum proves the existence of tuberculosis, but it is a comparatively late indication.

Prognosis.—In acute tuberculosis the outlook is unfavorable. In chronic tuberculosis the prognosis is dependent upon the stage of the disease, the constitutional vigor of the subject, and the hygienic conditions under which he is obliged to live. The mortality is high in young subjects (fifteen to twenty-five years) and those of feeble constitution. Unfavorable prognostic signs are persistent high temperature, acceleration of the pulse out of proportion to the fever, involvement of both lungs, continued indigestion, progressive loss of flesh, and the development of tuberculous lesions in other organs. The average duration is about three years.

Treatment.—*Prophylaxis.*—The means that may be instituted by the public authorities to limit the spread of tuberculosis include: The dissemination of information concerning the prevention of the disease; the supervision of schools, tenement-houses, factories, public conveyances, etc.; the systematic inspection by skilled veterinarians of dairies and slaughter-houses with the view of declaring unmarketable the milk and meat of tuberculous animals; the suppression of promiscuous expectoration in public places; the establishment of special hospitals and dispensaries for the indigent suffering from tuberculosis; and the compulsory registration of phthisical patients. Tuberculous patients should be taught to expectorate only into proper receptacles containing a disinfectant solution (5 per cent. carbolic acid) or into moistened rags or paper napkins, that should be burned before the sputum becomes dry. They should sleep alone. Their rooms should be sunny, well ventilated, and kept scrupulously clean.

Persons with a predisposition to tuberculosis can do much to increase their powers of resistance by strict attention to hygiene. Fresh air and sunlight, a healthy residence, an outdoor occupation, the wearing of warm clothes, with flannel next to the skin, and a diet of wholesome and nutritious food, temperate living, systematic exercise and daily cold sponging, followed by friction of the skin, are the factors to be relied upon in attempting to overcome individual susceptibility.

Sanatorium treatment undoubtedly gives the patient the best chance of recovery. In such institutions the patient spends in summer not less than nine or ten hours, and in winter not less than from six to nine hours, in the open air. The bedroom windows are kept open both winter and summer. He is given a mixed diet of wholesome food, and encouraged to eat as heartily as his digestive capacity will permit. If the disease is active, he is kept at absolute rest. For the most of the day he lies on a bamboo couch in the open air, warmth being maintained by abundant covering and, if necessary, by a hot stone at the feet. In quiescent tuberculosis moderate exercise is recommended, every precaution being taken, however, to guard against fatigue. To secure lasting improvement, the patient should remain in the sanatorium at least six months or a year.

Climatic Treatment.—To patients to whom a protracted stay in a sanatorium would be irksome or distasteful a change of climate offers the greatest hope of cure. As a rule, a high altitude should be selected; the atmosphere should be dry, and the temperature equable. Personal experience must decide the question of temperature; generally patients who feel better in summer will do well in a warm climate, and *vice versa*. The physician should have some knowledge of the locality, which should afford ordinary conveniences without being too crowded with sufferers similarly afflicted.

In selected cases a sea voyage is often very useful. According to Douglas Powell, it is most suitable to patients in the early stages, who have been previously healthy, who have

overworked nervous systems, and in whom the disease is more or less quiescent.

Treatment at Home.—This should be made to imitate as closely as circumstances will permit that which is followed in the sanatorium. The airiest and sunniest room should be selected for the patient. So long as he has fever absolute rest should be insisted upon. As much nourishing food should be allowed as he is capable of digesting.

Specific Treatment.—Tuberculin is a useful adjuvant to other measures in certain cases particularly those in which the general nutrition is good and the fever is slight. Contraindications are rapid emaciation, high temperature, active pleurisy, and nephritis. Hemoptysis and intercurrent infections call for suspension of the injections for a time. No matter which tuberculin is selected, the initial dose should be small— $\frac{1}{10000}$ mg. of old tuberculin (O. T.), tuberculin residue (T. R.) or broth filtrate (B. F.). The injections should be given at first once or twice a week, the dose being gradually increased, but never large enough to cause a reaction, not even a slight rise of temperature. The maximum dose varies with the individual. With O. T. it may be as high as 1000 mg. or as low as a few hundredths of a milligram. Of T. R. or B. F. the final dose may reach 20 mg. or more. If evidences of increasing sensitiveness appear the injections should be stopped for a time and then resumed with smaller doses.

Artificial Pneumothorax.—Compression and immobilization of the affected lung by the method of Forlanini, which consists in introducing warm nitrogen or oxygen into the pleural cavity has given good results in certain cases. It may be employed in moderately or far-advanced cases in which improvement has not occurred under ordinary methods of treatment, and in cases of uncontrollable hemorrhages. The chief contraindications are extensive involvement of both lungs, pleural adhesions or effusion, and serious complications, of any kind, especially cardiac or renal disease.

Medicinal Treatment.—If well tolerated and digested, cod-liver oil (1 to 4 fluidrams thrice daily) is of service in im-

proving the general nutrition. Creosote is useful if the expectoration is copious and purulent. The dose should be cautiously increased from 2 to 3 minims to 15 or 20 minims, three times a day. Alcohol is useful in some cases. Tonics, especially arsenic, are often serviceable.

Symptomatic Treatment.—Cough.—In many cases cough is indispensable and is best treated by promoting expectoration. For this purpose creosote, guaiacol carbonate and terpin hydrate are reliable remedies. Inhalations of creosote, compound tincture of benzoin, or terebene are often very effective. Local counterirritation is also of service. Rest in bed and regulation of diet are valuable aids. If the cough is very severe, sedatives must be given. Of these, the least objectionable are codein, heroin, hydrocyanic acid, and spirit of chloroform. Such combinations as the following will be found useful:

℞. Codeinæ sulphatis..... gr. vi-viiij
 Spiritus chloroformi..... fʒj
 Glycerin..... fʒj
 Succī limonis..... fʒss
 Aquæ..... q. s. ad fʒiij.—M.

SIG.—A teaspoonful as occasion demands.

℞. Codeinæ sulphatis..... gr. iv
 Acidi hydrocyanici diluti..... ℥ xxxij
 Syrupi tolutani..... q. s. ad fʒij.—M.

SIG.—A teaspoonful as required.

Hemoptysis.—Absolute rest is essential. An ice-bag may be placed over the suspected site of hemorrhage. Morphin, in doses of $\frac{1}{12}$ to $\frac{1}{8}$ grain hypodermically, is of value in controlling restlessness and irritative cough. Large doses, however, are objectionable, as they favor the retention of infected blood in the bronchi. Nitroglycerin has been recommended because it lowers the pressure in the pulmonary vessels. Saline purges seem to be of service when bleeding is slight but recurrent. Ergot and tannin are useless and so are inhalations of astringent vapors. Unless

collapse is imminent stimulants of all kinds should be avoided. Artificial pneumothorax should be considered favorably if hemorrhages persist and the site of the bleeding can be definitely determined.

Night-sweats.—Rest, a constant supply of fresh air, and regulation of diet often control the sweating. Sponging the body at bedtime with a solution of alum in alcohol and water or dusting it with a powder of tannoform (1 part) and zinc oxid (4 parts) is sometimes effective. The most reliable internal remedies are atropin ($\frac{1}{200}$ to $\frac{1}{120}$ grain), picrotoxin ($\frac{1}{80}$ to $\frac{1}{40}$ grain), and camphoric acid (5 to 10 grains).

Pyrexia.—In many cases the fever yields to absolute rest in bed or in a reclining chair, combined with life in the open air. Cold sponging is useful if the temperature is high. Antipyretic drugs are contraindicated.

Pleuritic Pains.—Mild attacks generally yield to sinapisms or the application of iodin. Strapping the affected side also affords relief. Severe pains should be treated by the application of dry cups or small blisters and the subcutaneous administration of morphin.

Diarrhea.—Diarrhea, the result of indigestion, usually yields promptly to restriction of the diet, rest, and the administration of a mild mercurial. Persistent diarrhea will demand the use of bismuth subnitrate (20 to 30 grains) combined with opium and intestinal antiseptics—salol, bismuth-beta-naphthol, or creosote. Combinations of tannigen (5 to 15 grains) or tannalbin (5 to 15 grains) with bismuth compounds are also useful:

R. Tannigen..... ʒj
 Bismuth-beta-naphthol..... ʒij
 Codeinæ sulphatis..... gr. iv.—M.

Fiant chartulæ No. xii.

SIG.—One every four hours.

ACUTE GENERAL TUBERCULOSIS

(Acute Miliary Tuberculosis)

Definition.—An acute infectious disease excited by the tubercle bacillus, and characterized anatomically by the presence of miliary tubercles in many parts of the body.

Etiology.—General tuberculosis is always secondary to a more or less obvious primary focus of tuberculosis somewhere in the body (lungs, lymph-nodes, joints, etc.). The infection is transmitted by the blood stream. The disease occurs most frequently in early adult life and certain acute infections, such as measles and pertussis, and wasting diseases predispose to it.

Pathology.—All the organs may be uniformly infiltrated with discrete tubercles, but more commonly certain organs, such as the brain and lungs, are more affected than others.

Symptoms.—The onset is gradual and characterized by anorexia, malaise, headache, increasing prostration, and fever. The temperature is moderately high (102° – 104° F.), very irregular, and marked by evening exacerbations and morning remissions. The respirations are hurried and the pulse is rapid (140 to 150) and feeble. Cough may or may not be present. Profuse sweating is common. As the disease advances typhoid symptoms frequently develop—dry, brown tongue, muttering delirium, subsultus tendinum, carphologia and stupor. Tubercle bacilli are sometimes found in the cerebrospinal fluid, but rarely in the sputum or blood. The leukocyte count is usually low.

In the so-called *pulmonary form* there are: Dyspnea, rapid breathing (30 to 50 a minute), hard cough, mucopurulent and bloody expectoration, and cyanosis. Signs of consolidation can rarely be elicited, but auscultation usually reveals dry and fine crackling râles.

In *meningeal infection*, there are: Intense headache, convulsive seizures, photophobia, delirium, ocular and facial palsies, stupor, coma, and Cheyne-Stokes breathing. Tubercles may occasionally be detected on the retina.

In infection of the *intestines and peritoneum* there are: Pain, tenderness, abdominal distention, and diarrhea.

Prognosis.—The disease is always fatal. The average duration is three or four weeks.

Diagnosis.—Acute miliary tuberculosis is not infrequently mistaken for *typhoid fever*, but the two diseases present the following differences:

TYPHOID FEVER	ACUTE GENERAL TUBERCULOSIS
Epistaxis is common.	Infrequent.
Temperature runs a regular course.	Temperature runs a very irregular course.
Diarrhea is frequent.	Infrequent.
A roseolar eruption is generally present.	Rarely present.
Respirations are rapid.	Usually much more rapid.
Pulse is rapid.	Usually much more rapid.
Cyanosis rarely marked.	Often distinct.
Facial palsies are absent.	Are not uncommon.
Widal reaction is present.	Is absent.
Tubercle bacilli are never present in the cerebrospinal fluid.	Are sometimes present.

Treatment.—This is purely palliative. The diet should consist of milk, eggs, and broths. Stimulants are required. Fever should be controlled by cold sponging or small doses of acetphenetidin. Severe cough and insomnia will call for morphin.

WHOOPING-COUGH

(Pertussis)

Definition.—An infectious disease, characterized by catarrh of the respiratory tract and peculiar paroxysms of cough ending in prolonged crowing or whooping inspiration.

Etiology.—Whooping-cough occurs largely in epidemics and is most frequently observed in young children, although unprotected adults are not entirely exempt. There is no doubt that the disease is contagious, especially in the catarrhal stage, that the infective agent is contained in the sputum, and

that the infection is transmitted chiefly, if not exclusively, by direct contact. The exciting cause of the disease is probably the Bordet-Gengou bacillus which is found in large numbers among the cilia of the epithelial cells lining the air passages. One attack usually confers permanent immunity.

Pathology.—There are no characteristic lesions. In fatal cases pulmonary complications such as bronchopneumonia, atelectasis, emphysema, etc., are usually present.

Symptoms.—There are three stages: (1) The catarrhal stage; (2) the paroxysmal stage; and (3) the stage of decline.

Catarrhal Stage.—The disease begins with the symptoms of coryza and bronchial catarrh—slight fever, sneezing, running from the nose, dry cough, and râles—but it does not respond to the ordinary remedies for catarrh, and after lasting one or two weeks passes into the paroxysmal stage.

Paroxysmal Stage.—The cough becomes more violent and paroxysmal. During the paroxysm the face is cyanosed, the eyes are injected, and the veins distended. The cough frequently induces vomiting, and, in severe cases, epistaxis or other hemorrhages. The close of the paroxysm is marked by a long-drawn, shrill, whooping inspiration due to spasm of the larynx.

The number of paroxysms, or “kinks,” varies from ten or twelve to forty or fifty in the twenty-four hours. From the forcible propulsion of the tongue against the lower incisors, an ulcer is occasionally formed on the frenum. The duration of this stage is three or four weeks.

Stage of Decline.—The paroxysms grow less frequent and less violent and finally cease. Protracted cases are followed by anemia and prostration.

During the entire course of the disease, which is usually from two to three months, a pronounced lymphocytic leukocytosis (15,000 to 30,000) is present.

Complications.—The chief complications are bronchopneumonia, acute emphysema, convulsions, and hemorrhage from the nose or into the conjunctiva. Paralysis from

meningeal hemorrhage occasionally occurs. Severe cases are sometimes followed by cancrum oris, chronic bronchitis, or tuberculosis.

Prognosis.—Owing to the frequent occurrence of serious complications whooping-cough must be regarded as a dangerous disease, especially in infants.

Treatment.—Prophylaxis consists in isolation of the patient and the thorough disinfection of all articles that have been used by him. Quarantine should last until the cough ceases.

Fresh air, sunlight, protection from changes of weather, and a light but nutritious diet are essential. In some cases it may be desirable to keep the patient in his room, or even in bed, for the first few days, but ordinarily, if the weather is good he need not be confined indoors. In advanced cases sea-air often acts most favorably.

Of the many special remedies advocated, those most worthy of confidence are belladonna (in ascending doses until constitutional effect is produced), antipyrin (about 1 grain for every year of the child's age, four times a day), quinin (10 grains a day at three years of age), and sodium bromid (3 to 5 grains every three hours at two years of age). Chloral (3 grains at two years of age) may be given in severe cases at bedtime to secure sleep. Such a combination as the following may be useful:

R. Sodii bromidi..... ʒiss
 Antipyrinæ..... ʒij
 Glycerini..... fʒij
 Aquæ menthæ piperitæ..... q. s. ad fʒiij.—M.

SIG.—A teaspoonful every three hours for a child of three years.

The exact value of vaccine treatment has not been demonstrated; good results have been reported, however, from injections into the buttocks every other day for 4 days of Bordet-Gengou bacilli, beginning with 1 billion and increasing to 6 billion.

Antiseptic and sedative sprays, when feasible, sometimes afford relief; the best are compound tincture of benzoin,

menthol (5 per cent. in liquid paraffin), and resorcin (1 per cent. aqueous solution). An elastic abdominal belt is of some value in controlling the vomiting.

The child must be carefully guarded during convalescence, on account of the danger of catarrhal pneumonia. Tonics, especially quinin, iron, and cod-liver oil, are very useful at this period.

CHOLERA

(Asiatic Cholera; Epidemic Cholera)

Definition.—An acute infectious disease, generally epidemic, caused by *Spirillum cholera asiaticæ* and characterized by vomiting, excessive serous purging, painful cramps, and collapse.

Etiology.—Cholera is endemic in certain parts of India, and from time to time spreads into western countries. The exciting cause is the *Spirillum cholera asiaticæ* of Koch, a short, slightly curved, motile rod with a single flagellum. This organism is found abundantly in the intestinal discharges of choleraic patients, but not in the blood.

The disease always spreads along the lines of traffic, hence epidemics nearly always begin at the sea-coast and extend inland. Cholera is slightly, if at all, contagious. As in the case of typhoid fever human being contract the disease by ingesting the germs, usually in water or on food. Flies are undoubtedly important factors in disseminating the infection to food. Laundresses and nurses, from contact with the evacuations, often acquire the disease. Epidemics are more frequent in summer than in winter. No age is exempt, but the aged are more susceptible than the young. The intemperate, the debilitated, and those suffering with gastro-intestinal catarrh are especially predisposed.

Pathology.—The body is shriveled; movements of the corpse are sometimes observed; rigor mortis is marked and prolonged. The tissues are dry, and the large veins and right side of the heart contain thick, dark blood. The serous

cavities are empty and their surfaces sticky. The intestines contain more or less rice-water fluid, from which cultures of bacilli can be made.

The mucous membrane has a pinkish color and is often the seat of ecchymoses; the solitary and Peyer's glands are swollen. Frequently, extensive desquamation of the epithelial lining is observed. The liver and kidneys are the seat of acute parenchymatous degeneration.

The symptoms of cholera are due to the absorption of a poison (endotoxin). Apparently normal epithelium prevents the absorption of the endotoxin contained in the bodies of the bacilli and set free by their destruction.

Symptoms.—The *period of incubation* is from two to five days. The severity of the symptoms varies considerably. In well-marked but favorable cases there are three stages: (1) Stage of invasion; (2) stage of collapse; (3) stage of reaction.

Stage of Invasion.—The disease usually begins with malaise, headache, diarrhea, rumbling noises in the intestines, and colic. Frequently these symptoms continue a few days and then subside; such cases are termed *cholérine*, and are as infectious as the fully developed disease.

Stage of Collapse.—The diarrhea grows more marked; the evacuations become copious, lose their feculent character, acquire a rice-water appearance, and are discharged forcibly but without pain. Vomiting soon develops, and the ejected material resembles that passed by the bowels. Thirst is unquenchable. Severe cramps seize the muscles of the calves of the legs, thighs, arms, and abdomen. The surface is cold and covered with a clammy sweat; the breath is cool; the temperature in the axilla ranges from 95° to 85° F., while in the rectum it may rise to 103° F. or more. The voice is husky and finally reduced to a whisper; the respirations are quickened; the pulse becomes more and more feeble; the body is livid and shriveled; the features are pinched and sometimes distorted; the eyes are frightfully sunken. The urine is more or less suppressed, and the little that is passed fre-

quently contains albumin and sugar. Consciousness is usually retained until near the end, when coma sets in.

The duration of this stage is from a few hours to two days.

Stage of Reaction.—In this stage the symptoms gradually ameliorate, the stools become less frequent, the temperature returns to normal, the urine increases in amount and convalescence is soon established.

Occasionally, instead of convalescence, symptoms of a typhoid type develop (*cholera typhoid*), such as moderate fever, a dry brown tongue, muttering delirium, and coma. This condition, which is usually fatal, is probably the result of a secondary infection or of uremia.

Cholera Sicca.—This term has been applied to cases in which the intoxication is overwhelming and the patient dies within a few hours after the onset, even before the occurrence of diarrhea.

Complications and Sequelæ.—The chief complications are: nephritis, pneumonia, pleurisy, parotitis, ulceration of the cornea, croupous inflammation of the throat and fauces, abscesses, and local gangrene.

Diagnosis.—The differential diagnosis between Asiatic cholera and *cholera morbus* has already been considered (see p. 75).

Prognosis.—This depends largely upon the type. The mortality averages about 50 per cent. In the aged, very young, debilitated, and intemperate the disease is very fatal. In individual cases early collapse and a low surface temperature are unfavorable conditions. Most deaths occur within forty-eight hours after the onset.

Treatment.—Personal prophylactic measures against the disease include removal from the infected districts, restriction of the diet to bland, easily digested food, avoidance of uncooked vegetables, thorough sterilization of drinking-water and milk, the protection of all food from contamination by flies and other insects, the avoidance of overwork, exposure to wet and cold, and undue excitement, and the prompt

treatment of any gastro-intestinal disturbance that may arise. Certain acids, especially sulphuric acid, have long been advocated as preventives of cholera. Finally, vaccination with attenuated cholera cultures, as practiced by Haffkine in India, has given encouraging results.

Precautionary measures pertaining to the sick comprise isolation, absolute cleanliness, and the thorough disinfection of excreta, soiled clothing, etc.

The medicinal treatment of cholera resolves itself into that of the prodromal stage, that of the algid stage, and that of the reaction stage.

Prodromal Stage.—From the first appearance of diarrhea the patient should go to bed and remain there. Food should be withheld. If there be a history of indigestible food having been taken, a laxative dose of calomel should be given; otherwise, aperients should be avoided. Hot stupes may be applied to the abdomen. Morphin is better avoided. For the diarrhea, bismuth subnitrate is perhaps the best astringent. Rogers highly recommends potassium permanganate (two 2-grain salol-coated pills every one-half hour) with $\frac{1}{100}$ grain of atropin, hypodermically, morning and evening.

Algid Stage.—Intravenous injections of warm saline solutions undoubtedly afford the best means of combating the anhydremia and of restoring the failing circulation. Rectal injections of hot tannic solutions (2 per cent.), as strongly recommended by Cantani, may also be used. The body-temperature should be maintained by hot applications or hot baths. Diffusible stimulants, such as ether and camphor, may be given hypodermically.

To allay thirst, ice or iced Seltzer water may be given at frequent intervals. The painful cramps are best treated by warm applications, hot baths, gentle friction with anodyne liniments, and, above all, by intermittent chloroform inhalations. In suppression of urine the most promising measures are dry cupping over the loins and rectal and intravenous injections of saline solution.

Reaction Stage.—In this stage liquid foods in small quantities are permissible. Milk with lime-water, whey, thin gruels, albumin-water, and light broths are the most appropriate. The return to ordinary food should be effected very gradually.

DYSENTERY

The term dysentery is applied to a number of conditions characterized by abdominal pain, frequent diarrheal discharges containing mucus and blood, and tenesmus. Anatomically, there may be catarrhal, ulcerative or pseudo-membranous changes in the intestine, especially in the colon. The excitants of dysentery may be chemical irritants, various bacteria, or certain protozoa (*Ameba coli*, *Balantidium coli*). Two specific varieties are recognized: (1) Bacillary, and (2) amebic.

BACILLARY DYSENTERY

Etiology.—Bacillary dysentery is caused by the *Bacillus dysenteriae*, described by Shiga, Flexner, Kruse, and others, and of which there are several strains. The disease is especially prevalent in Japan, the Philippines, and other tropical countries, but it frequently occurs elsewhere both sporadically and in epidemics. As in typhoid fever, the infection is transmitted chiefly by contaminated food and drink. Doubtless flies often aid in its dissemination. Overcrowding and unsanitary conditions in general favor its spread, hence it has prevailed extensively at times in camps, jails, etc. Lowered vitality from any cause is a predisposing factor, and in consequence dysentery is not an uncommon secondary or terminal event in many wasting diseases. The summer diarrheas of children may also be associated with the dysentery bacillus.

Pathology.—The disease is usually acute, but it may be chronic. The changes predominate in the large bowel and in acute cases usually consist of hyperemia, swelling of the mucosa, enlargement of the solitary follicles, and superficial erosions. The lymph-glands are often enlarged. In some cases false membrane is formed on the surface of the bowel

(*diphtheritic dysentery*), and in others more or less widespread gangrene occurs. In chronic cases the mucous membrane is thickened and reddened and shows superficial erosions, but rarely any pronounced ulceration.

Symptoms.—The disease begins with colic and diarrhea. Within twenty-four hours tenesmus develops and the stools become mucous and bloody. In some cases shreds of false membrane are passed. The temperature rises usually to between 101° and 103° F., the tongue is coated, the appetite is lost, the abdomen is tender and somewhat excavated, and vomiting sometimes supervenes. Weakness and emaciation rapidly develop. Toxic symptoms—muscular pains, delirium and stupor—are not uncommon in serious infections. The blood serum yields an agglutination reaction with dysentery bacilli of the type isolated from the stools. In fatal cases death usually occurs after two or three weeks, but it may occur within three or four days. In favorable cases the duration is from ten days to three or four weeks. Occasionally the disease loses its acute character and continues with remissions and exacerbations for an indefinite period.

Complications.—Peritonitis, usually circumscribed and due to extension of the inflammation through the bowel, is not uncommon. Inflammation of other serous membranes, arthritis, peripheral neuritis, and parotitis are sometimes observed.

Diagnosis.—The diagnosis of dysentery can usually be made without difficulty from a study of the general symptoms. The determination of the exact nature of the infection, however, must rest upon the serum agglutination tests and the isolation of the bacillus from the stools. In *acute catarrhal enteritis* tenesmus is absent; the stools are not bloody and mucous, nor are they so frequent or so small. *Intussusception* may be distinguished by the abrupt onset, persistent vomiting, sausage-shaped abdominal tumor, and absence of fever, at least at the outset.

Prognosis.—This depends upon the severity of the attack and the resistance of the individual. The more marked the

toxic symptoms are the more unfavorable is the outlook. In some epidemics the mortality has reached 40 per cent.

Treatment.—The prophylactic measures adopted in typhoid fever and cholera are applicable in dysentery. Absolute rest in bed is imperative. In acute cases the diet should consist of milk diluted with barley-water, peptonized milk, infant foods, egg albumin, chicken broth, and milk toast. In chronic cases, soft boiled eggs, pulled bread, steamed rice, oysters, tender meats, wine-jelly, and custard may be allowed. An unirritating purgative (magnesium sulphate, calomel, or castor oil) is indicated at the onset. Subsequently opium should be given to inhibit peristalsis and to relieve the tenesmus. It may be given hypodermically in the form of morphin, or by the stomach or bowel. Turpentine stupes or sinapisms over the abdomen are advisable. Tenesmus may also be relieved by ice suppositories, iodoform suppositories, injections of warm mucilage of starch (1 ounce) or of cocain solution (10 minims of a 4 per cent. solution), or irrigations with warm normal saline solution. In the chronic form irrigations with solutions of silver nitrate (10 to 20 grains to the pint) are effective. These should be given every day, the fluid being introduced very gently by means of a fountain syringe. Internally, bismuth subnitrate (30 to 40 grains) with antiseptics, as salol (5 grains) or benzonaphthol (5 grains), is useful. Considerable benefit may also be derived from serum therapy. The serum should be polyvalent or, if feasible, one corresponding to the type of infection present. From 50 to 100 c.c. may be given every six to twelve hours. For collapse, stimulants and subcutaneous injections of saline solution may be used.

AMEBIC DYSENTERY

Etiology.—This form of dysentery prevails chiefly in the tropics, but sporadic cases and even small epidemics not very rarely occur in temperate regions. The exciting cause of the disease is the ameba coli (*Entameba histolytica*), a unicellular

protoplasmic mass about 15 to 30 μ in diameter, containing a nucleus and one or more vacuoles. The infection is usually transmitted through drinking water or food, and encysted amebæ in the stools of convalescents or healthy carriers, rather than the motile organisms in dysenteric stools, are the chief factors in spreading the disease.

Pathology.—The lesions are found mainly in the large intestine. At first the mucous membrane is hyperemic and edematous. Later localized areas of cellular infiltration appear in the form of hemispheric elevations and these ultimately break down, leaving deep ulcers with infiltrated undermined edges. In advanced cases the whole intestinal wall is much thickened. The amebæ are found in the dejections, and at the base of the ulcers, in the adjacent lymph-vessels, and sometimes in the blood-vessels, whence they are frequently (20 per cent. of fatal cases) conveyed to the liver where they cause colliquative necrosis and the so-called amebic abscess.

Symptoms.—Mild attacks are not rarely observed, in which for weeks or months the only symptoms are indigestion, colicky pains, and slight diarrhea. The diagnosis rests upon the discovery of the parasites in the stools. Recovery is the rule, but the condition may become acute or may slowly pass into a chronic phase. In other cases the infection is acute, and marked by fever, abdominal pain, vomiting, tenesmus, mucus and blood discharges, great weakness and emaciation. Death may result from exhaustion, or the condition may gradually become chronic. Irrespective of the mode of onset, the disease shows a marked tendency to assume a chronic form. It is then characterized by continuous or intermittent diarrhea; the passage of mucus, blood, and perhaps pus in the stools; more or less abdominal discomfort; afternoon fever; and, ultimately, marked anemia and wasting. Tenesmus may not be pronounced. Uncomplicated cases usually yield to treatment, but relapses are extremely common. Death is most frequently due to *hepatic* or *hepatopulmonary abscess*. Other *complications* are perforation of the bowel, peritonitis from extension of

the ulcerating process, intestinal hemorrhage, and cicatricial stenosis of the bowel.

Diagnosis.—Unlike the amebic form, *bacillary dysentery* is usually acute, is rarely associated with pronounced ulceration of the bowel or hepatic abscess, and is frequently productive of a general intoxication. *Syphilis* and *carcinoma of the rectum* must always be considered in the differential diagnosis.

Treatment.—The same general measures should be followed as are adopted in bacillary dysentery. *Ipecacuanha* has been shown to exert a specific effect and should always be given a thorough trial. The drug may be given in pills heavily coated with salol (40 grains the first night and reduced 10 grains each night until only 10 grains are given) or, better, in the form of emetin hydrochlorid ($\frac{1}{2}$ grain in 20 minims of saline solution, twice a day, subcutaneously, for ten or twelve days) or of emetin-bismuth iodid (2 to 3 grains in salol-coated pills each night for 12 nights). Irrigation of the bowel is efficacious. It should be done once a day by means of a fountain-syringe, the tube being inserted high up and the fluid (a quart to two quarts) retained, is possible, for fifteen minutes. The best solutions are, perhaps, quinin (1 to 5000 gradually increased to 1 to 1000) and silver nitrate (1 to 2000 gradually increased to 1 to 500). If the rectum is very irritable, it is advisable to inject a small quantity of cocain solution (4 per cent.) before introducing the irrigator. In intractable cases recourse may be had to colostomy or appendicostomy and irrigation of the colon through the wound.

TETANUS

(Lockjaw)

Definition.—An acute infectious disease excited by a special bacillus, and characterized by painful tonic spasms of the voluntary muscles.

Etiology.—The exciting cause is the *Bacillus tetani*, a motile, spore-bearing, anaërobic rod, multiplying in garden-earth, street dirt, and the intestinal discharges of herbivorous ani-

mals. The disease is contracted through contamination of wounds with matters containing the bacillus or its spores. Lacerated and punctured wounds about the soles of the feet and palms of the hands are especially liable to become infected. Occasionally no history of injury is obtainable. The colored race appears to be particularly vulnerable.

Pathology.—There are no characteristic lesions. Congestion of the spinal cord and of the nerves leading to the wound is sometimes seen. The bacillus produces at the point of inoculation a toxin, which ascends the peripheral nerves to the spinal cord where it becomes firmly united to the nerve tissues and excites a strychnin-like action.

Symptoms.—The *period of incubation* is usually from a few days to two or three weeks. The disease begins with a feeling of rigidity in the muscles of the neck and lower jaw; by degrees the muscles of the back, trunk, and lower extremities are similarly involved. The brow is wrinkled, the corners of the mouth are drawn upward (*risus sardonicus*), the jaws are tightly closed (*trismus*), and the body becomes arched, the patient resting on his head and heels (*opisthotonos*). There is extreme hyperesthesia, so that the slightest touch causes a violent exacerbation of the spasm, which is attended by excruciating pain. If the respiratory muscles are involved, there is intense dyspnea. The temperature is variable. It is usually elevated during the paroxysms and just before death it may rise to 107° F. or more. The mind is clear to the end. The duration is from a few days to several weeks.

Diagnosis.—In *strychnin-poisoning* there is no history of a primary wound, the onset is sudden, the convulsions are general from the beginning and are intermittent, and the course is very short. In *tetany* the spasms are limited to the extremities and larynx, and are intermittent. In *hysteria* there is no history of a primary wound, trismus is absent, and other characteristic nervous phenomena are present.

Prognosis.—In acute cases the prognosis is very grave, death usually resulting within a week from heart-failure,

asthenia, or asphyxia. Cases developing after a long period of incubation (more than ten days) and not characterized by violent seizures not infrequently end in recovery. The average mortality is about 60 per cent.

Treatment.—The most important safeguard against the occurrence of tetanus is the thorough cleansing and disinfection of all wounds. When there is ground for suspicion that tetanus may develop, as in wounds contaminated by garden earth, street dirt, or stable refuse, the local treatment should be supplemented by prophylactic injections of tetanus antitoxin. From 500 to 1000 U. S. A. units should be given subcutaneously and repeated once a week until the wound heals. In the fully developed disease antitoxin is of much less value, but it should always be used. From 10,000 to 25,000 units should be given intramuscularly or subcutaneously, and, unless there is definite improvement, a similar amount should be given again in from eighteen to twenty-four hours. At the same time at least 3000 units should be given intraspinally, after the removal of a requisite amount of cerebrospinal fluid. Intravenous injections may also be used, although there is some danger of anaphylaxis. The drugs most effective in subduing the convulsions are the bromids and chloral. These should be given in large doses. Morphin is a useful adjuvant. Inhalations of chloroform afford temporary relief. The patient should be kept absolutely quiet and protected from cold. The administration of nutriment in liberal quantities (by nasal tube if necessary) is of the utmost importance. Continuous enteroclysis is often advisable.

EPIDEMIC POLIOMYELITIS

(Acute Anterior Poliomyelitis; Acute Infantile Spinal Paralysis)

Definition.—An acute infectious disease, occurring chiefly of young children, characterized anatomically by inflammation in the gray matter of the spinal cord with destruction of the nerve-cells in the anterior horns, and manifested clinically by

febrile disturbances and rapid atrophic paralysis of various muscles.

Etiology.—The disease may be sporadic or epidemic. It is more prevalent in cold than in warm countries. Most cases occur in the summer months. Children below the age of six years are especially susceptible, but adults may be attacked. The affection is contagious, though probably not highly so, and doubtless is often transmitted by healthy persons ("carriers") who have been in contact with the sick. The researches of Flexner and Noguchi have demonstrated that the causative agent is a minute anaërobic organism and that the infection atrium is the nasal mucosa.

Pathology.—The changes are most frequently found in the lumbar segments of the cord and are especially pronounced in the anterior horns of gray matter, but not rarely the medulla, pons, and cerebrum are also affected. The meninges often share in the inflammatory process. In early cases microscopic examination reveals distention of the blood-vessels, cellular infiltration, and degenerative changes in the ganglion cells. In long-standing cases the lesions consist in atrophy of the anterior horns, loss of ganglion cells, and overgrowth of neuroglia.

The motor nerve-fibers corresponding to the diseased segments and the muscles innervated by those fibers are also the seat of degenerative changes.

Symptoms.—The onset is marked by moderate fever (101° – 103° F.), restlessness, headache, pain in the back and limbs, and muscular soreness. Less frequently there is vomiting or diarrhea, and occasionally convulsions occur. The mind is usually clear. In the course of a day or two a flaccid paralysis makes its appearance, all four extremities and the trunk, the lower extremities, one limb, or, perhaps, only a group of muscles being involved. The legs are especially prone to suffer. The paralysis reaches its maximum in a few hours or days, and then begins to improve, in many cases only a small amount remaining at the end of a few weeks or months. Complete recovery is sometimes observed. The constitu-

tional symptoms soon pass off; sensation remains intact; the sphincters are not disturbed, and there is no tendency to bed-sores. The muscles showing decided paralysis lose their tendon reflexes, rapidly waste, and soon yield the reaction of degeneration. Permanent deformity often ensues from the retardation of growth in the paralyzed members and the overcontraction of the unantagonized muscles.

Varieties.—Apart from the common *spinal* form, there are (1) *abortive* cases, in which the constitutional disturbance is unattended by paralysis and complete recovery occurs in a few days; (2) *meningitic* cases, in which the early symptoms simulate closely those of epidemic cerebrospinal meningitis; (3) *bulbar* cases, in which the nuclear centers in the medulla oblongata are involved; and *polyneuritic* cases in which pain in the limbs and general hyperesthesia are for a few days the most conspicuous features.

Diagnosis.—Except in epidemics the diagnosis is not possible before the appearance of paralysis. In *multiple neuritis* the paralysis develops more gradually, is more marked in the distal than in proximal parts of the limbs, and is symmetric, and, moreover, the sensory disturbances are more lasting. *Myelitis* may be distinguished by the presence of anesthesia, paralysis of the bladder and rectum, and the tendency to bed-sores. The early occurrence of flaccid paralysis and the absence of cocci in the cerebrospinal fluid will distinguish the meningitic type from *epidemic cerebrospinal meningitis*. The *cerebral paralyzes of childhood* are spastic, attended by exaggerated reflexes, and not followed by rapid wasting.

Prognosis.—Unless the initial symptoms are very severe or the muscles of respiration are affected, the prognosis as regards life is good. The death-rate has ranged between 5 and 30 per cent. In all cases much of the paralysis disappears, and occasionally the improvement is so marked that the usefulness of the members is not seriously impaired.

Treatment.—During the acute stage the patient should be isolated and confined to bed. Mild laxatives and febrifuges

may be used with some advantage. Hexamethylenamin (2 or 3 grains, every two hours during the acute stage) has been recommended by Dana and others for the purpose of sterilizing the cerebrospinal fluid, but it is of doubtful value. Aspirin or morphin may be necessary for the relief of pain. Warm baths and lumbar puncture are also worthy of trial. The affected limbs should be wrapped in cotton-wool and kept in such a position that the paralyzed muscles will not be over-stretched. In the course of three or four weeks, if the acute features have entirely subsided, mechanical treatment (massage, passive movements, electricity) should be instituted. The treatment of advanced cases is chiefly surgical, and has for its object the prevention or correction of deformities.

EPIDEMIC ENCEPHALITIS

(Lethargic Encephalitis)

Definition.—A subacute, infectious, and mildly contagious disease, characterized anatomically by foci of inflammation in the central nervous system, especially in the regions of the brain stem and basal ganglions, and clinically by a diversity of symptoms, but most commonly by fever, somnolence, ophthalmoparesis, and certain hyperkinetic phenomena, such as muscular twitchings, tremors and choreiform movements.

Etiology.—The disease occurs in epidemics, which are usually small, which are prone to follow outbreaks of influenza, and which prevail more frequently in winter and spring than in summer. No age is exempt, but the majority of cases occur between twenty and fifty. The inciting agent has not been isolated, but the virus exists in the nasopharynx and is filterable.

Pathology.—Macroscopic changes, other than slight congestion of the cerebral meninges, are usually wanting. Microscopically, the important feature is the presence of cellular collections, chiefly lymphocytic, about the blood-vessels. Degenerative changes in the ganglion cells, while never entirely absent, are relatively slight.

Symptoms.—The onset is, as a rule, acute and marked by chill, headache, lassitude, generalized pains, catarrh of the upper respiratory tract, and moderate fever, which lasts from a few days to two or three weeks. The most constant symptoms of the full-developed disease are somnolence or profound lethargy, ophthalmoparesis (diplopia, ptosis, limitation of ocular movements,) and certain extra-pyramidal motor disturbances, such as twitching of the abdominal muscles, coarse tremors of one or both arms, or choreic movements of the face and limbs. A masklike facial expression is also common.

Delirium often supervenes and somewhat frequently there is persistent insomnia instead of somnolence. Facial palsy, disturbances of respiration and deglutition, hiccup, spasticity of the extremities, facial neuralgia and pains in the proximal portion of the limbs ("central" pains), and secretory derangements, particularly excessive sweating and sialorrhea, are sometimes observed. Meningitic symptoms (rigidity of the neck, Kernig's sign, etc.), general convulsions, paralysis of the arms or legs, and anesthesia are exceptional. Moderate leucocytosis is the rule and examination of the spinal fluid usually shows slight lymphocytosis (20 to 100 cells per cm.), sugar and globulin increase, and a luetic gold curve.

Duration and Prognosis.—The duration varies from a few days to several months. The mortality is apparently between 20 and 35 per cent. Recovery may be complete, but in a large proportion of cases there is permanent disability, important sequels being tremors with muscular spasticity (Parkinsonian syndrome), chronic chorea, mental deficiency, a change in personality, pronounced neurasthenia, psychic disturbances suggesting dementia præcox or general paresis, and adiposis with polyuria.

Treatment.—Rest in bed, protection from excitement, an abundance of liquid or semiliquid food, and careful nursing are first in importance. Removal of 10 to 20 c.c. of spinal fluid at intervals of a few days is sometimes of service. Such sedatives as bromids, barbital and scopolamin (hyoscin) are often required.

DENGUE

(Breakbone Fever; Dandy Fever)

Definition.—An acute infectious disease, characterized by pains in the muscles and joints, a variable rash, and a febrile course of two paroxysms.

Etiology.—Dengue is confined almost entirely to hot climates. Although it occurs in epidemics, it is probably not contagious, but transmitted through infected mosquitoes.

Symptoms.—The *period of incubation* is from three to five days. The invasion is usually sudden, and is attended with lassitude, chilliness, headache, intense pain in the muscles and joints, and high fever. The latter rises rapidly, often reaching a maximum of 104° to 105° F. in a few hours. The skin and conjunctivæ are congested; the pulse is accelerated (100–130); the urine is scanty; the superficial lymph-glands are enlarged; and the joints are painful, tender, and slightly swollen; mild delirium is sometimes noted. In three or four days sweating occurs; the temperature falls; and the pains abate, leaving the patient comparatively comfortable, but extremely weak. This remission lasts one or two days, and is followed by a second paroxysm, in which all the original symptoms return, though with less severity and for a shorter period. A roseolar eruption usually accompanies the second elevation of temperature. It lasts one or two days and is followed by desquamation.

Complications are rare and recovery is the rule. Hemorrhages from the mucous membranes occasionally occur. Convalescence is often slow, the soreness in the joints persisting for a long time.

Diagnosis.—*Acute Rheumatism.*—This disease runs a more protracted course and lacks the paroxysmal character and the eruption of dengue.

Treatment.—There is no specific remedy. A mercurial aperient should be given at the onset. The pains are best

relieved by acetphenetidin, salicylates, and morphin. The diet should be liquid and sustaining.

HYDROPHOBIA

(Rabies ; Lyssa)

Definition.—A specific infectious disease of certain carnivorous animals, especially dogs and wolves, communicated to man by direct inoculation, and characterized by slight fever, intense spasm of the muscles of the throat, delirium, paralysis, and coma.

Etiology.—Rabies invariably results from the bite of a rabid animal, usually a dog. In the animal the disease is characterized by depression of spirits, loss of appetite, followed by excitement, aimless roving, a morbid desire to bite, and finally by paralysis and death from exhaustion. The poison is contained in the central nervous system and secretions, especially the saliva. Bites on the face and on exposed parts are particularly liable to be followed by infection.

Pathology.—The bacteriology is obscure. Microscopically, accumulations of embryonic cells are found in the cord and bulb surrounding the motor nerve-cells or displacing them (rabid tubercles of Babes). Still more important from a diagnostic standpoint, hyaline bodies, probably protozoa, are found in the cells of the cerebral cortex, cerebellar cortex, horn of Ammon, and spinal ganglia (Negri bodies).

Symptoms.—The *period of incubation* is usually from three weeks to three months, rarely as long as twelve months. The *onset* is characterized by slight fever, anxiety, depression, restlessness, and pain in the wound or cicatrix. In about a day symptoms of the *convulsive stage* appear. These consist in great difficulty in swallowing, severe clonic spasm of the laryngeal muscles, salivation, general hyperesthesia, tetanoid seizures, hallucinatory delirium, and prostration. Anything that excites the swallowing reflex, such as the sight of water, may bring on the painful spasm of the throat muscles. In the

course of two or three days, if the patient does not die from exhaustion or heart-failure, the *paralytic stage* supervenes, in which the convulsions and delirium give way to ascending paralysis and unconsciousness.

Diagnosis.—An hysterical condition, known as *pseudo-hydrophobia*, is not uncommonly observed in neurotic persons who have been bitten by non-rabic dogs. It may simulate true hydrophobia, but there is no fever, the symptoms last longer than those of rabies, and the patient often barks like a dog, tries to bite, and becomes very emotional.

Prognosis.—Once developed, the disease is invariably fatal.

Treatment.—Suspicious bites should be thoroughly disinfected and cauterized with caustic potash or strong carbolic acid and treated as open wounds. As soon as possible the patient should be given the benefit of Pasteur's immunizing treatment, which consists in a series of inoculations with properly prepared spinal cords from artificially infected rabbits. The mortality from rabies among patients promptly immunized is less than 1 per cent. The treatment of the attack is purely palliative. An attempt should be made to maintain nutrition by rectal alimentation and to control the convulsive paroxysms by injections of morphin and inhalations of chloroform.

LEPROSY

(*Lepra*; Elephantiasis Græcorum)

Definition.—A chronic infectious disease caused by the *Bacillus lepræ*, and characterized by granulomatous formations in various parts of the body, more particularly in the skin, mucous membranes, and nerves.

Etiology.—Leprosy exists to a greater or less extent in all parts of the world, but it is especially prevalent in India, China, Japan, and other oriental countries. In the United States the chief foci are in Louisiana, Florida, and Minnesota. The disease is only mildly contagious. The exciting cause is the *Bacillus lepræ*, which resembles the tubercle bacillus and which

is found in the specific lesions and in the secretions of the nose and mouth.

Pathology.—The bacillus excites a chronic proliferative reaction, resulting in the formation of a granuloma, somewhat like that observed in tuberculosis and syphilis. Caseation, however, does not occur, but necrosis of the individual cells ensues and slowly advances to the stage of ulceration.

Symptoms.—The *period of incubation* may be but a few weeks or as long as three or four years. Prodromal symptoms, consisting of headache, malaise, irregular attacks of fever, and pains in the joints, are sometimes observed. Two clinical forms are recognized, the nodular (tubercular) and the anesthetic, but very often the manifestations are mixed. The *nodular type* is characterized by the development of large or small nodular elevations of a dusky-red color in various parts of the body, especially on the face, the extensor surfaces of the elbows and knees, and the hands. After a time these break down, forming ulcers which do not readily heal, or they are transformed into dense cicatricial tissue, causing unsightly deformities. The mucous membranes of the eyes, nose, and throat may also be involved. The *anesthetic type* is manifested by lancinating pains and areas of hyperesthesia, followed by the appearance of yellowish macules, which spread peripherally and eventually become anesthetic. Sooner or later trophic changes occur. These comprise milk-white patches (*lepra alba*), areas of brown pigmentation, bullous eruptions, loss of hair, muscular atrophy, mutilating ulcers, and a disappearance of the phalanges one by one, through disintegration and absorption (*lepra mutilans*). In advanced cases the skin of the face is irregularly thickened and nodular (*leontiasis*), the ears are leathery, the facial hair is lost, the eyelids are everted, and the hands and feet are crippled and deformed. The diagnosis is made certain by the detection of the lepra bacilli in an excised nodule or in the nasal secretion. The disease is very chronic, often lasting twenty years or more, and occasionally it is arrested.

Treatment.—Segregation is advisable in all cases. The patient's general nutrition should be improved by wholesome, digestible food, personal cleanliness, frequent warm baths, and tonics. Of the many special remedies recommended, chaulmoogra oil (doses of 5 minims, very gradually increased, according to gastric tolerance, to 1 dram, three times a day) seems to be the only one possessing real merit. Recently, even better results have been reported from weekly intramuscular injections of the ethyl esters of the fatty acids contained in chaulmoogra oil.

PLAGUE

Definition.—Plague is an acute infectious disease, excited by the *Bacillus pestis bubonicæ*, characterized by high fever, extreme prostration, inflammation of the lymph-glands (buboes), and often by pustules, pneumonia, and hemorrhages.

Etiology.—The plague bacillus, which is a short, encapsulated, non-motile, non-spore-bearing organism, is found in all the fluids and organs of the infected body. Transmission of the disease may occur directly through the entrance of infected material into some abrasion of the skin or a mucous membrane, but in the large majority of cases it probably occurs indirectly through the bite of a rat-flea, this insect conveying the infection from the rat, an animal particularly susceptible to plague. The disease may occur in any climate and in any season, but it flourishes best in the tropics and in warm weather. Persons of any age may be attacked. Overcrowding and bad sanitary conditions favor outbreaks.

Pathology.—Three forms of buboes may be recognized: *Primary buboes*, which develop near the point of inoculation and which are characterized by marked periglandular edema; *secondary buboes*, which occur in the immediate neighborhood of the primary lesion and are produced by infection through the lymph vessels; and *tertiary buboes*, which occur in all parts of the body and are caused by infection from the blood. Suppuration rarely occurs in the last two varieties, but is

common in primary buboes. The skin, mucous membranes, and serous membranes are often studded with punctate hemorrhages. The lungs are congested and frequently show areas of bronchopneumonia. The heart is flabby and dilated. The spleen is enlarged and congested. The liver and kidneys are swollen, hyperemic, and show cloudy swelling.

Varieties.—Four forms are generally described: bubonic, pneumonic, septicemic, and abortive or ambulatory.

Symptoms.—The *period of incubation* varies from two to ten days.

Bubonic Plague.—The onset is usually sudden, with chilliness, headache, general pains, vomiting, and great prostration. The fever is high and irregular. The general appearance is often suggestive; the eyes are bright, the expression is anxious, the nostrils are dilated, the body is flexed. The buboes usually appear early or within a few hours of the onset, and are most frequent in the groin, femoral region or axilla. Suppuration takes place in the second week and is not rarely followed by gangrene. The pulse is frequent and feeble, the respirations are hurried, and the urine is scanty. Delirium is common. Pustules sometimes appear on the body and in many cases multiple hemorrhages are noted. In the majority of cases death occurs before the buboes suppurate.

Pneumonic Plague.—In this form severe pneumonia, usually of the lobular type, is the important feature. The general symptoms are similar to those of bubonic plague, but primary buboes are usually absent. Death occurs, as a rule, within the first week.

Septicemic Plague.—In this comparatively rare form no primary point of infection can be detected. However, all the lymph-glands are more or less enlarged, the systemic disturbance is profound, and death almost invariably results in from one to three or four days.

Abortive or Ambulatory Plague.—In this form one or more buboes frequently develop, but do not go on to suppuration, and the general symptoms are mild.

Prognosis.—This depends upon the severity of the epidemic, the type of disease, and the intensity of the infection in the individual case. The death-rate varies from 25 to 95 per cent.

Prophylaxis and Treatment.—The most important safeguards against the spread of plague are isolation of the sick, sick, rigid quarantine, thorough disinfection of the patient's discharges and all articles that may have become soiled with the discharges, and good general sanitation, including the prevention of overcrowding, the proper disposal of excreta and garbage, and the destruction of rats and their haunts. Inoculation with sterilized cultures of plague bacilli (Haffkine's vaccine) is also worthy of trial. Treatment of the disease is chiefly symptomatic. Antiplague serum seems to have some curative power.

GLANDULAR FEVER

This is an infectious, probably contagious, disease of infancy and childhood, characterized by chilliness, headache, general malaise, moderate fever, slight reddening of the throat and tonsils, and pronounced inflammatory swelling of the lymph-nodes, most frequently of the deep cervical nodes, posterior to or just beneath the sternocleidomastoid. The glands rarely suppurate and complications are few, although nephritis has been noted. The duration varies from ten days to four weeks. The treatment is essentially symptomatic.

ACUTE INFECTIOUS JAUNDICE

(Weil's Disease; Spirochætosis Icterohæmorrhagica)

These names are applied to an acute infectious disease, commonly epidemic, excited by a spirochete—*Leptospira icterohæmorrhagiæ*—and characterized by a sudden onset, irregular fever (102° – 104° F.), generalized pains, nausea, vomiting, and jaundice, which appears about the fourth day. The spleen is usually enlarged, the urine is often albuminous, polymorphonuclear leucocytosis is almost always present, hemorrhages, especially from the nose and bowel, are common, delirium sometimes supervenes and in unfavorable cases the clinical picture becomes that of acute yellow atrophy of the liver. The acute symptoms last two or three weeks and convalescence is usually slow.

The disease, which is apparently transmitted through the urine of infected rats, is most common in Japan and Egypt, but it has frequently been reported from Europe. The mortality has not usually exceeded 5 per

cent., although in Japan it has been as high as 38 per cent. The epidemic jaundice occurring in the United States seems to be communicable by direct contact, shows a predilection for children, and is apparently not of spirochetal origin, although its etiology is not definitely known.

ROCKY MOUNTAIN SPOTTED FEVER

This is an acute infectious disease occurring in certain valleys of the Rocky Mountains and transmitted by the bite of a tick. The inciting agent is not definitely known, but it is suspected to be a Rickettsia organism similar to that occurring in typhus fever. The attack begins with a chill, severe pains in the head, back and limbs, and fever. The temperature rises rapidly, and may reach 104° F. at the end of a week. In the milder cases it subsides gradually, reaching normal at the beginning or end of the third week. The conjunctivæ are injected, the pulse is very rapid (120-140), the bowels are constipated, and in many cases there is marked jaundice. About the third or fourth day a red macular rash appears on the wrists and ankles, and then spreads over the body. In the course of a few days the macules become definitely petechial. Gangrene of the skin in certain parts and general edema are sometimes observed in severe cases. The average mortality in some districts (Bitter Root Valley) is 90 per cent., but in Idaho it is less than 5 per cent. Death occurs most frequently within the first ten days. The treatment is altogether symptomatic.

KALA-AZAR

(Visceral Leishmaniasis; Tropical Splenomegaly; Dum-dum Fever)

Kala-azar is a chronic, specific, infectious disease, prevailing especially in India and certain other regions of the Orient. It is caused by the *Leishmania donovani*, a round or oval body about the size of a blood platelet, which is probably conveyed by the bed-bug or some other biting insect.

The disease begins with an irregular remittent fever (100°-103° F.), leucopenia (4000-2000), and rapid enlargement of the liver and spleen, especially of the latter. The fever usually subsides in from two to six weeks, but recurrences alternating with periods of apyrexia are the rule. As the disease progresses marked weakness, emaciation, and anemia supervene, the skin acquires a grayish color, and general edema frequently develops. The spleen extends below the navel in three-fourths of the advanced cases. The mortality is said to range between 75 and 95 per cent. In addition to general hygienic measures, quinin in very large doses and tartar emetic ($\frac{1}{2}$ to $1\frac{1}{2}$ grains in 1 per cent. solution, intravenously, every other day until 25 or 30 grains in all have been given) are of definite value.

MALTA FEVER

(Mediterranean Fever)

Definition.—An infectious disease of long duration, excited by the *Micrococcus melitensis* and characterized by periods of fever, alternating with periods of normal temperature, profuse sweats, neuralgic pains, progressive weakness, and anemia.

Etiology.—While the disease is especially prevalent in Malta, it has frequently been reported from other tropical and subtropical countries. It has been proved conclusively that the large majority of cases are due to the use of milk from infected goats. Infection through contaminated food other than goats' milk, through the inhalation of dust containing the micrococci, or through the bites of insects appears to be comparatively rare. The organism is found in the blood and leaves the body in the urine, feces, and milk. The period of incubation is about two weeks.

Symptoms.—The most striking symptom is the fever. This usually rises gradually to a maximum of 103° or 104° F., runs an irregular remittent course for from one to five weeks, and then gradually drops to normal, where it remains several days, when a relapse occurs. This sequence of events is repeated again and again, the duration of the disease varying from eight weeks to a year or more. During the pyrexial periods there are general depression, profuse sweating, neuralgic pains, especially in the legs, and often swelling of the joints. The spleen is always enlarged; the blood contains the *Micrococcus melitensis* and specific agglutinins; and as the disease progresses anemia and debility develop.

Complications.—Bronchitis, peripheral neuritis, orchitis, and arthritis are the most common complications. Hyperpyrexia is sometimes observed.

Prognosis and Treatment.—The prognosis is good, both as regards life and ultimate recovery. The mortality is between 2 and 3 per cent. The treatment is entirely symptomatic. Hydrotherapy is the most useful measure.

SLEEPING SICKNESS

(Trypanosomiasis)

Definition.—An infectious disease, endemic on the west coast of Africa, excited by the *Trypanosoma gambiense*, and characterized by swelling of the lymph-glands, moderate fever, progressive weakness and emaciation, increasing lethargy, and, finally, death in coma.

Etiology and Pathology.—The cause of sleeping sickness is the *Trypanosoma gambiense*, a minute, flagellated, motile protozoon, which gains entrance to the body through the bite of a tsetse-fly (*Glossina palpalis*).

The organism is found in the lymph-glands, blood, and cerebrospinal fluid. The symptoms of the disease are apparently due to an accumulation of lymphocytes in the perivascular spaces of the brain and spinal cord, secondary degeneration of the nerve-cells, and overgrowth of neuroglia (chronic meningo-encephalomyelitis).

Symptoms.—The course of the disease is extremely slow. In the first stage, which lasts from a few months to three or four years, the only symptoms are enlargement of the lymph-glands and the presence of trypanosomes in the blood and lymphatic fluids. The second stage usually lasts several months and is marked by the appearance of the parasites in the cerebrospinal fluid, increasing weakness and lethargy, a peculiar apathetic expression, a feeble monotonous voice, tremor of the hands, a shuffling gait, an evening rise of temperature (101° to 102° F.), a rapid feeble pulse, lymphocytosis, and, finally, coma. During the last two or three weeks the temperature is usually subnormal.

Prognosis and Treatment.—Recovery rarely if ever occurs after the appearance of trypanosomes in the cerebrospinal fluid. Death results from the disease itself or from some intercurrent affection, such as pneumonia or septic meningitis. Treatment is of little avail. Atoxy-intramuscularly and tartar emetic intravenously have been used, but without much success.

WORM INFECTION—HELMINTHIASIS

TAPEWORM OR CESTODE INFECTION

Varieties.—*Tænia solium*; *Tænia saginata*; *Dibothriocephalus latus*; *Hymenolepis nana*; *Tænia echinococcus*.

***Tænia Solium* (*Pork-measle Tapeworm*).**—This worm exists in the larval state (*Cysticercus cellulosæ*) in the hog, which becomes infected by eating food contaminated with the eggs of the parasite derived from human feces. Man becomes infected by eating raw or rare pork containing the larvæ. The mature worm is from 6 to 12 feet in length. The head has a double row of hooklets, and is attached to the body by a thread-like neck. The uterus in the gravid segments has from 7 to 10 lateral branches on each side of its median stem. When ova gain access to the stomach of man through the pylorus, as in vomiting, or by the ingestion of contaminated food, they may produce embryos which pass through the walls of the alimentary canal, effect a lodgment in the muscles or organs, and there develop into bladderlike larvæ (*cysticerci*). This parasite is rare in America.

***Tænia Saginata* (*Fat Tapeworm*).**—The encysted form occurs in the flesh of the ox (*Cysticercus bovis*), which becomes infected by eating food containing larvæ derived from human feces. Man becomes infected by eating undercooked or undercured beef. The mature worm is from 12 to 25 feet in length. The head is without hooklets, and the median stem of the uterus gives off on each side from 15 to 35 slender branches. *Tænia*

saginata is the most common tapeworm in North America and certain parts of Europe.

Dibothriocephalus Latus (*Broad Tapeworm*).—The larval form of this parasite is found in certain fresh-water fish. Man is infected by eating uncooked fish. The mature worm is of yellowish-brown color, and is from 25 to 40 feet in length. The head is flat and without hooklets; the segments are usually broader than long; the sexual opening is in the middle of the flat surface of the segment, and the uterus is in the form of a roset. This worm is common in Northern Europe, but rare in America.

Hymenolepis Nana (*Dwarf Tapeworm*).—This parasite develops without any intermediate host. Man is infected probably through food contaminated with eggs derived from the feces of infected rats or other human beings. The adult worm is only an inch or an inch and a half long. The head is small and provided with a single circle of hooklets. Great numbers are usually present. The geographic distribution of the parasite is extensive.

Tænia Echinococcus.—The adult worm occurs in the intestine of the dog. In man the parasite appears only in the larval state (see Hydatid Cysts of the Liver).

Symptoms.—In many cases of cestode infection there are no subjective symptoms. Some patients, however, present digestive disturbances, bulimia, colicky pains, anal pruritus, emaciation, anemia, and certain reflex phenomena—vertigo, palpitation, itching of the nose, and choreiform movements. The diagnosis rests on the detection of segments or eggs in the stools. Infection with *Dibothriocephalus latus* may be the cause of severe anemia, which is attributed to a toxin produced by the parasite.

Treatment.—By way of preparatory treatment it is advisable to restrict the diet for a day or two to liquids, and to empty the bowel as completely as possible by saline purges. The best teniafuges are oleoresin of aspidium (1 dram), pelletierin (5 to 10 grains), and pumpkin seed (2 to 4 ounces).

R̄.	Oleoresinæ aspidii.....	f3j
	Pulveris acaciæ.....	3ij
	Syrupi zingiberis.....	3iss
	Aquæ.....	q.s. ad f3j

Misce et fiat Emulsum

Sig.—To be taken at one dose.

A full dose of Epsom salts or compound jalap powder should be given a few hours after the teniafuge. The treatment is successful only when the head of the worm is passed.

ROUND-WORM OR NEMATODE INFECTION

Ascaris Lumbricoides (*Eel-worm or Common Round-worm*).

Eel-worms develop from eggs which have entered the digestive tract through contaminated water or food. Apparently no intermediate host is required. They are of a grayish or pinkish color, and in form resemble earthworms. They live in the small intestine, but not rarely, especially in febrile conditions, they migrate, entering the stomach, bile-ducts, respiratory tract, or urinary passages, or perforating into the abdominal cavity through an intestinal ulcer.

Symptoms.—Symptoms are often absent. Sometimes there are dyspepsia, voracious appetite, colicky pains, mucous stools, anal pruritus, anemia, and reflex nervous phenomena—night-terrors, grinding of the teeth, disturbances of vision, choreiform movements, and convulsions.

Treatment.—Santonin ($\frac{1}{4}$ to $\frac{1}{2}$ grain); wormseed oil (10 minims in capsule or on sugar); and fluidextract of spigelia (1 to 2 fluidrams) and efficient remedies. The anthelmintic should be followed by a purge.

R̄.	Santonini.....	gr. vj
	Hydrargyri chloridi mitis.....	gr. vj
	Sacchari.....	gr. xxiv.—M.

Fiant chartulæ No. xii.

SIG.—One powder morning and evening (Starr).

Oxyuris Vermicularis (*Pin-worm; Seat-worm*).—Pin-worms are small white worms, measuring from one-eighth to one-half

inch in length. They are most frequent in children, but may occur at any age. Infection occurs directly through the eggs, which are conveyed to the mouth on the fingers or enter the stomach through contaminated food. The adult worms are most numerous in the cecum. The females migrate to the rectum, where they deposit their eggs. Intense itching at the anus, especially at night, is the most common symptom. The disturbances described under common round-worm infection may also occur.

Treatment.—Copious injections of the infusion of quassia, lime-water, or of salt and water (1 dram to 1 pint) are useful in removing the females from the rectum. To rid the small intestine of the young parasites santonin and calomel should be given by the mouth. Care should be taken to prevent auto-infection.

Necator Americanus and Ankylostoma Duodenale.—These are hookworms of the family Strongylidæ. The former prevails in the Americas (southern part of the United States, West Indies, South America), and the latter in Europe, Asia, and Africa. The male parasite is about $\frac{1}{3}$ of an inch long and has an expanded tail; the female is about $\frac{1}{2}$ inch long and has a pointed tail. Hookworms inhabit the duodenum and jejunum.

Ankylostomiasis is a common disease in tropical and subtropical countries. In temperate regions it prevails chiefly among miners. The eggs of the parasite escaping with the feces bring forth embryos which in a few days develop into larvæ. The latter may enter the digestive tract directly through food or water, but it has been shown that in the majority of cases entrance to the bowel is effected indirectly by way of the skin, the route being from the blood to the heart, from the heart to the lungs, from the lungs to the larynx, and thence to the esophagus, stomach, and bowel. The entrance of the larvæ through the skin, usually that of the feet or legs, is followed by an eruption known as "ground itch."

Symptoms.—Anemia, more or less severe, weakness, and emaciation are the chief symptoms. Eosinophilia is usually present. Digestive disturbances and mental lassitude are frequently observed. The diagnosis depends upon the detection of the eggs or the adult worms in the stools.

Treatment.—Thymol (30 grains, repeated in two hours) and oil of chenopodium (8 minims at 7, 8, and 9 o'clock in the morning) are the best remedies. A saline purge should be given before and after the anthelmintic, and when thymol is used solvents of the drug, such as alcohol, oil, butter, etc., should be avoided on the day of treatment.

Trichina Spiralis.—This is a minute worm derived from the hog. Man is infected by eating raw or rare pork containing encysted larvæ. The latter pass into the small intestine, where they develop into adult worms. The females penetrate the lymphatic spaces, and there bring forth young, which are carried by the lymph or blood to the muscles, where they develop into encysted larvæ. Trichinous capsules, impregnated with lime, are visible to the naked eye. The hog acquires trichiniasis by eating scraps of infected pork or infected rats.

Symptoms of Trichiniasis (Trichinosis).—No decided symptoms develop unless the infection is severe. In well-marked cases gastro-intestinal disturbances appear on the second or third day. These disturbances consist in colicky pains, nausea, vomiting, and serous diarrhea.

In from one to two weeks symptoms of an acute myositis develop. These include severe muscular pain and soreness, edema, especially of the face, profuse sweating, and remittent or intermittent fever. Hoarseness from involvement of the larynx and dyspnea from involvement of the diaphragm are often noted. Eosinophilia (10 to 50 per cent.) is almost constantly present. Delirium and stupor are not uncommon in severe infections. Trichinæ are sometimes found in the blood and spinal fluid. At times the symptoms closely resemble

those of typhoid fever. In favorable cases recovery ensues in from three to eight weeks.

Diagnosis.—*Typhoid Fever.*—The history, the presence of eosinophilia, of intense muscular soreness, of edema, of parasites in the stools or in a fragment of muscle removed from the arm, and the absence of a typical rash and of the Widal reaction will lead to a correct diagnosis.

Prognosis.—This depends upon the number of worms ingested. Early diarrhea is favorable. The mortality ranges from 5 to 30 per cent.

Treatment.—The most efficient prophylactic measure is the thorough cooking of all pork products. In the first stage cathartics are indicated. Anthelmintics—santonin and thymol—have been recommended. After migration, the indications are to relieve pain by means of opiates, hot baths, and warm embrocations, and to support the strength by concentrated liquid diet and stimulants.

Filaria Bancrofti (Filaria Sanguinis Hominis).—This is a small, thread-like worm, chiefly met with in warm climates. The adult occupies the lymphatics, and the female brings forth actively motile larvæ, which find their way into the circulating blood. It is a curious fact that the larvæ are found in the blood chiefly at night. The medium of infection is the mosquito, in the body of which the larvæ reach a certain stage of development before being again transmitted to man.

Symptoms.—These are not always present, but chyluria, lymph scrotum, lymph varices, and elephantiasis are not uncommon manifestations.

Treatment.—Thymol and methylene-blue have been recommended, but they are of doubtful utility.

Filaria Loa (Filaria Diurna).—This is a common parasite on the west coast of Africa. The worm occupies the connective tissues throughout the body and often approaches the surface near the eye, causing an edematous swelling with itching, and, at times, more or less intense conjunctivitis. The treatment consists in removing the parasite under local anesthesia.

DISORDERS OF METABOLISM AND FOOD DEFICIENCY DISEASES

GOUT

(Podagra)

Definition.—A disturbance of metabolism involving the disposition of purin substances in the blood, characterized in its typical form by deposits of monosodium urate in the joints and other structures, and by recurrent attacks of acute arthritis.

Etiology.—Gout most frequently develops in the fourth and fifth decades. It is more common in males than in females. It is often hereditary. The excessive use of wines or malt liquors, overeating, sedentary habits, nervous strain, and chronic lead-poisoning are predisposing factors.

Pathology.—The pathology of gout is still obscure. It is generally conceded that the disease is in some way associated with excess of uric acid compounds in the blood; but whether these compounds are the sole cause of the constitutional disturbances, and whether the excess in the blood is due to increased formation or diminished excretion, or both, are questions that await solution.

The only distinctive anatomic lesions of gout are those of the joints. These consist of deposits of sodium biurate (tophi) in the cartilages and fibrous tissues and secondary inflammatory changes. In long-continued cases the joints become irregularly enlarged and stiff. Ultimately ulceration of the superficial tissues may ensue, with the discharge of the

uratic concretion. The small joints of the feet and hands are usually the first to be affected, but subsequently other joints, like those of the ankles, wrists, and elbows, become involved. Uratic deposits are often found also along the tendons, in the external ear, in the nose, and in various other parts.

In *acute* cases the affected joint, most frequently the metatarsophalangeal of the great toe, is intensely hyperemic, swollen, and edematous.

Chronic interstitial nephritis, arteriosclerosis, and hypertrophy of the heart are important concomitant lesions.

Clinical Varieties.—(1) Articular gout, which may be acute or chronic; (2) non-articular or irregular gout.

SYMPTOMS.—**Acute Gout.**—The attack is usually preceded by certain prodromes—restlessness, insomnia, moroseness, irritability, dyspepsia, and changes in the urine, this secretion becoming scanty, high-colored, and deficient in urates. The arthritic phenomena usually appear suddenly in the early morning hours, and are characterized by pain and swelling in the ball of the great toe. The affected joint is so tender that the slightest pressure causes agony. It is of a reddish-purple color; its surface is glazed; and the overlying veins are full and distinct. During the paroxysm the temperature is moderately elevated (101° – 102° F.) and the pulse quickened. Toward daylight the pain subsides to a great extent and the patient falls asleep. During the day he is comparatively comfortable, but there are severe exacerbations for several successive nights. At first the attacks may be a year apart, but as they multiply the interval grows less, until finally the patient is seldom entirely free from suffering.

Retrocedent Gout.—This term is applied to a condition in which the arthritic phenomena suddenly subside and grave gastric, cardiac, or cerebral symptoms follow.

Chronic Gout.—The joints are affected one by one, and become stiff, irregularly enlarged, and deformed. Chalk-stones, or tophi, sometimes ulcerate their way through the

skin and are discharged. Similar deposits are frequently found along the tendons and in the helix of the ear.

Constitutional symptoms similar to those occurring in non-articular or irregular gout are more or less conspicuous.

Non-articular Gout (Uric Acid Diathesis; Latent Gout; Goutiness; Lithemia).—This form of gout is more often met with in America than the articular variety. It presents the following clinical features:

Gastro-intestinal Phenomena.—The tongue is usually coated and the breath heavy; the appetite is variable, sometimes it is lost, at other times it is inordinate; acid eructations, heart-burn, and flatulence are frequent gastric symptoms.

Urinary Phenomena.—The urine is scanty, high-colored, of high specific gravity (1025 to 1035), and on standing throws down an abundant brick-dust sediment. The solids render the urine irritating, so that dull aching in the loins and burning in the penis after micturition are common symptoms. A trace of sugar is sometimes detected on chemical examination. The urine often stains the clothes red.

Circulatory Phenomena.—These consist in increased arterial tension, accentuation of the second aortic sound, and a tendency to arteriosclerosis.

The Blood.—An increase of uric acid in the blood above the normal of 3 mg. per 100 c.c., with the patient on a purin-free diet, is confirmatory evidence in favor of gout, although it is by no means pathognomonic.

Nervous Phenomena.—These are extremely varied, and include headache, disturbed sleep, tinnitus aurium, depression of spirits, impairment of memory, loss of energy, irritability, and neuralgic pain in various parts of the body.

Complications and Sequelæ.—These include: Chronic interstitial nephritis, arteriosclerosis, hypertrophy of the heart, angina pectoris, apoplexy, chronic bronchitis, and certain cutaneous affections—chronic eczema, urticaria, and psoriasis.

Diagnosis.—*Acute Rheumatism.*—This more commonly affects the larger joints; it is markedly migratory; it is associ-

ated with higher fever and more copious perspiration; and it shows far greater tendency to endocardial and pericardial inflammations.

Rheumatoid Arthritis.—The family history and habits of the patient, the involvement of joints rarely attacked in gout (temporomaxillary, thumb, cervical vertebræ, sternoclavicular), the peculiar deformities, the special joint changes (atrophy or newformation of bone), the absence of tophi and of an excess of uric acid in the blood, and the muscular atrophy will serve to distinguish rheumatoid arthritis from gout.

Prognosis.—Acute gout rarely proves fatal; recurrence, however, is to be expected. On account of the tendency to arterial and renal complications, the prognosis of chronic gout, when the disease is fairly established, should be somewhat guarded.

Treatment.—*The Acute Attack.*—The best remedies are colchicum and cinchophen (atophan). From 20 to 30 minims of the tincture of colchicum may be given every three hours until the pain is relieved or looseness of the bowels occurs, or 7 grains of cinchophen may be given every three or four hours. The alkalis are useful adjuvants. The free use of water should be encouraged. Constipation should be relieved by a full dose of blue-mass or a saline draft. Opium or acetphenetidin may be required for the relief of pain. The affected part should be elevated and wrapped in cotton-wool, or covered with warm fomentations or with cloths soaked in magnesium sulphate solution. The diet should be light and non-stimulating.

Chronic Gout.—As regards diet, simplicity and moderation are of the utmost importance. Generally speaking, a diet composed for the most part of milk, farinaceous foods, succulent vegetables and eggs is most suitable. The foods most likely to disagree are veal, liver, sweetbreads, hashes, croquettes, concentrated soups, vegetables rich in nucleins (peas and beans) pastry, sweets, coffee, malt liquors, and heavy wines. Some patients are exceedingly intolerant of acid fruit.

Water-drinking between meals should be encouraged. No more should be eaten than is absolutely necessary to satisfy

hunger. The patient should be warmly clothed and should avoid as far as possible exposure to sudden atmospheric changes. Systematic exercise in the open air is very beneficial. If active exercise is not feasible, massage may be strongly recommended. All overwork of mind should be forbidden. Hydrotherapy—tepid sponge-baths and douches—is useful. Heavy, robust patients often derive much benefit from the Turkish bath. Visits to certain mineral springs—Bedford, Saratoga, Harrowgate, Carlsbad, Contrexeville, Aix-les-Bains—sometimes have a very salutary effect.

Free action of the bowels should be secured. The occasional use of calomel or blue-mass at night, with a saline in the morning, is often of value. Among the special remedies advocated for gout may be mentioned colchicum, cinchophen, alkalis, alkaline mineral waters, arsenic, and iodids. Colchicum and cinchophen are most effective in the acute paroxysms, although small doses with alkalis may be of benefit in the intervals. The prolonged use of arsenic in small doses seems to be of some value. Iodids are sometimes of service in relieving the concomitants and sequels of gout, but have little, if any, effect upon the disease itself. Salicylates relieve pain, but are distinctly inferior to colchicum.

Chronic affections of the joints are best treated by gentle massage, friction, and warm sulphur baths.

RICKETS

(Rachitis)

Definition.—A constitutional disease of early childhood, characterized chiefly by defective nutrition of the osseous structures.

Etiology.—Rickets is most common between the sixth month and the third year of life. Poor nutrition of the mother, bad air, lack of sunlight, and, above all, defective feeding, are important etiological factors, hence the disease is observed most frequently in large cities and among the children of the poor.

Pathology.—The changes indicate tardy bone-formation with undue preparation for it. They consist of marked increase of cartilage at the ends of the bone, excessive production of osteoid tissue by the medullary and periosteal structures, irregular and deficient calcification, and resorption of bone from within out of proportion to that formed by the periosteum. In consequence of these defects the bones are softened, irregularly enlarged, especially in the region of the epiphyses, and often bent or otherwise distorted. With the arrest of the disease which usually occurs spontaneously after the third year, true ossification of the osteoid tissue ensues and thus the deformities already produced are to a greater or less extent perpetuated. In addition to the osseous changes, enlargement of the liver, spleen, and mesenteric lymph nodes is often observed.

Symptoms.—*General Features.*—These comprise excessive perspiration, especially about the head; restlessness at night; disinclination to move or to be moved, owing to general hyperesthesia; delayed or irregular dentition; gastro-intestinal disturbances; pallor, emaciation and a flabby state of the muscles; and undue protuberance of the abdomen (“pot-belly”) owing to muscular atony, flatulence and enlargement of the liver and spleen.

Skeletal Features.—The head is relatively large and often more or less square; the fontanelles remain open longer than normal and the sutures are wide; parts of the occipital and parietal bones may be abnormally thin and yielding (cranio-tabes); enlargements or beads are often noted at the junction of the osseous and cartilaginous portions of the ribs (rachitic rosary), the sides of the chest may be flattened and the sternum thrust forward (pigeon-breast); a horizontal furrow (Harrison’s groove) is sometimes seen where the thorax joins the belly; curvatures of the spine frequently occur; epiphyseal enlargements are observed in the long bones; bending of the leg bones with the production of bow-legs or knock-knees is common; finally, the pelvic outlet may be very much narrowed.

Complications.—Green-stick fractures, bronchopneumonia, convulsions, involuntary micturition, tetany, and laryngismus stridulus sometimes occur.

Prognosis.—Rickets is not in itself a fatal disease, and under appropriate treatment may disappear within a few months.

Prognosis.—Improvement of the hygienic conditions, especially an abundance of fresh air and sunshine, and regulation of the diet are the most important parts of the treatment. If dependence must be had upon artificial feeding, fresh cow's milk, properly modified to suit the digestive power of the infant, should be recommended. Egg-albumin and fresh meat juice are sometimes useful additions. Cod-liver oil (30 to 40 minims) thrice daily, after meals at the age of one year) and phosphorus (1 to 1½ drops of phosphorated oil, thrice daily, after meals at the age of one year) are the most efficient drugs.

R̄. Emulsi olei morrhuæ. f℥ iv
 Olei phosphorati. f℥ i-f℥ iss.—M.
 Sig.—A teaspoonful after meals.

DIABETES MELLITUS

Definition.—A disorder of metabolism characterized by impairment of the ability of the tissues to utilize carbohydrate, and manifested by persistent hyperglycemia, glycosuria, and ultimately acid intoxication.

Etiology.—The disease is most common between the ages of thirty and sixty. Hebrews appear to be especially prone to it. Males are more frequently affected than females. Over-eating and sedentary habits favor its occurrence, and therefore it is much more prevalent among the well-to-do than among the poor. Hereditary influence may be traced in some cases. Among determining causes may be mentioned mental strain, acute infections, and injury of the brain or spinal cord.

Pathology.—The underlying cause of diabetes is somewhat obscure. It is generally conceded, however, that a lesion of the pancreas involving the islands of Langerhans is chiefly concerned in the process and that the essential feature of the

disease—the inability to utilize sugar—is caused by a loss of an internal secretion of the pancreas. Possibly, other glands with which the pancreas in relation—adrenal, hypophysis, and thyroid—may at times also be involved in the disturbance of carbohydrate metabolism. The effect of lesions of the central nervous system, such as puncture of the floor of the fourth ventricle, seems to be merely to cause an excessive mobilization of sugar by the liver. In addition to disease of the pancreas, there is usually more or less enlargement of the liver, and in more than one-half of the fatal cases chronic nephritis is also present.

Failure of the tissues to utilize carbohydrate results in an excess of glucose in the blood (hyperglycemia), and to this feature is attributable the glycosuria, thirst, polyuria, emaciation, and many other symptoms of the disease. A secondary result of the disordered carbohydrate metabolism is the loss of ability to oxidize completely the fats, leading to the appearance of acetone bodies in the urine (ketonuria), first acetone, then diacetic acid, and later in severe cases, betaoxybutyric acid.

Symptoms.—*General Phenomena.*—The onset is usually insidious, but it may be abrupt. As a rule, the first symptoms to attract attention are weakness, excessive thirst, frequent micturition, and polyuria. Pruritus, especially of the genitalia, may be an early manifestation. The appetite may or may not be inordinate. In severe cases emaciation is marked. The skin is dry and harsh; the saliva is scanty; the tongue is frequently red and glazed; the teeth readily decay; and the bowels are usually constipated. Impotence is common; the knee-jerks are diminished or lost, and many patients complain of neuralgic pains and muscular cramps. The most important nervous phenomenon, however, is coma, which is the direct cause of the fatal issue in more than one-half of the cases. It is due to acidosis (β -oxybutyric acid), and frequently develops as the result of overexertion, intercurrent disease, or nervous shock, but it may occur as the last event without any

definite exciting cause. It is often preceded by headache and somnolence, and is accompanied by air-hunger (long-drawn inspirations), a fruity odor to the breath, digestive disturbances, and symptoms of collapse. It is almost always fatal in from a few hours to a few days.

The Urine.—The urine is increased in amount, the daily output varying from 3 or 4 liters to 10 liters or more; it is pale in color, of increased specific gravity (1025–1040), and contains sugar, and frequently one, two or all three of the acetone bodies. The sugar content varies from 0.5 to 10 per cent., and the daily output from 50 to 1000 grams. Albumin and casts are also present in many cases. The total nitrogen and ammonia are increased, the latter often to 8 grams or more (normally from 0.5 gram to 1.5 grams). The excess of alkali is required for the neutralization of the organic acids and the amount of ammonia excreted is therefore an approximate measure of the degree of acidosis, 2 grams corresponding to about 6 grams of β -oxybutyric acid and 5 grams to about 20 grams of β -oxybutyric acid. The amount of urine and the sugar and acid contents usually diminish markedly before the onset of coma.

The Blood.—Hyperglycemia is a constant feature, the concentration of sugar often rising to 0.15 or 0.5 per cent. (normally 0.06 to 0.1 per cent.). As the amount of sugar excreted depends to great extent upon the degree of renal permeability, estimation of the blood-sugar provides a much more reliable criterion of the severity of the disease than estimation of the urinary sugar.

Complications.—The most common complications result from diminished resistance to bacterial infection and comprise boils, carbuncles, tuberculosis, and pneumonia. The last is not rarely followed by gangrene of the lung. Skin eruptions, especially eczema, are frequently observed. Nephritis occurs in at least one-half of the cases. Peripheral neuritis is common. Defective vision from cataract, retinitis or optic neuritis may occur. Gangrene of the extremities, usually as a

result of obliterating endarteritis, occurs with considerable frequency.

Diagnosis.—Recognition of the disease in its early stages is only possible through routine examination of the urine. Transient glycosurias and other forms of mellituria (lactosuria, pentosuria, etc.), must be excluded. In alimentary glycosuria the administration of 100 grams of glucose two hours after a light breakfast may be followed by the appearance of sugar in the urine, but the whole amount is less than 2 grams and the excretion never persists more than six hours. Glycosuria on a diet containing starch but no sugar should be regarded as *prima facie* evidence of diabetes.

Prognosis.—This depends upon the severity of the disease and the faithfulness with which appropriate treatment is carried out. The outlook is, as a rule, more favorable in obese, middle-aged persons than in thin, young subjects. In children the disease is quickly fatal. Generally speaking, those are mild cases in which 3 ounces of bread (about 60 grams of starch) can be metabolized without the occurrence of glycosuria; those are moderately severe cases in which glycosuria occurs with this amount of carbohydrate but disappears with a strict meat-fat diet; and those are severe cases in which glycosuria still persists with a carbohydrate-free diet. The duration of the disease varies from several weeks in the very acute cases to twenty years or more in the very chronic ones.

Treatment.—This is chiefly *dietetic*. The indications are to maintain nutrition, to increase the tolerance for carbohydrates, to lessen hyperglycemia, and to prevent or diminish acidosis. In mild cases, after a few days of strict diet,¹ the

¹ **Articles Permitted.**—*Animal Foods.*—Meats of all kinds (except liver), fish (except oysters and clams), eggs, and soups without flour.

Fats.—Butter, lard, olive oil, cheeses of all kinds, and cream (small amounts).

Vegetables.—Celery, lettuce, cucumbers, cresses, asparagus, egg-plant, spinach, string beans, tomatoes, cauliflower, cabbage, squash, mushrooms and pickles.

patient may be permitted to take three-fourths of the carbohydrate that he is able to tolerate. In moderately severe cases he may be permitted to take two-thirds of his ascertained tolerance, but one day in seven his diet should be restricted to 5 per cent. vegetables and one-half the usual quantity of protein and fat. In severe cases the "starvation" diet of Allen is sometimes remarkably successful. This consists in almost complete fasting, giving only water, clear broth, and, if necessary, a small amount of whisky, for about three days, or until the urine has become sugar-free. After the fasting a small amount (150 grams) of 5 per cent. vegetables (lettuce, cucumbers, spinach, asparagus, celery, tomatoes, cabbage, string beans) is allowed, and then the quantity of carbohydrate is very cautiously increased by the addition of 10 per cent. vegetables (onions, turnip, squash, carrots, mushrooms), 15 per cent. vegetables (green peas, parsnips, canned lima beans), and 5 per cent. fruits (grape fruit, ripe olives) and 10 per cent. fruits (oranges, strawberries, peaches, blackberries, watermelon, pineapple), until sugar reappears or the tolerance reaches 3 grams of carbohydrate per kilogram of body weight. At the same time the protein is gradually increased from 20 grams (3 eggs) until the patient is receiving 1 gram per kilogram of body weight, or if carbohydrate tolerance is zero, only $\frac{3}{4}$ gram per kilogram of body weight, and the fat is cautiously added and increased until the patient ceases to lose weight or receives in the total diet 20 to 30 calories per kilogram of body weight.

Desserts.—Custards and ice-cream containing no starch and sweetened with saccharin.

Beverages.—Tea, coffee or lemonade sweetened with saccharin, mineral waters, whiskey, brandy, Rhine wine or Burgundy.

Articles Prohibited in Strict Diet.—Bread, cake, biscuit, and pastry of all kinds (most gluten breads contain a considerable amount of starch and are indigestible); fruits of all kinds; cereals of all kinds; macaroni, potatoes, carrots, beans, beets, turnips, corn, peas, and parsnips; milk, cocoa, and chocolate; malt liquors, cider, sweet wines, and liqueurs; preserves and ice-cream.

Woodyatt, Newburgh and others have recently shown that a diet of 900 calories, derived chiefly from fat, is as effective in rendering the patient sugar free and in reducing basal metabolism as starvation, and, moreover, that it is less dangerous to the patient and much less of a hardship. The diet advocated by them, and which often yields good results, even in severe cases, is one of low carbohydrate, low protein and high fat (2 grams per kilogram of body weight).

General hygienic measures are of great importance, especially the prevention of mental and physical overexertion, worry and excitement. A moderate amount of physical exercise is beneficial in the milder cases, but considerable rest is necessary in the severe forms of the disease.

Drugs have not proved of much value in diabetes. Recently, however, Macleod has prepared a pancreatic extract, freed from digestive enzymes and tentatively termed "insulin," which inhibits the hyperglycemia in animals resulting from extirpation of the pancreas, piqûre of the fourth ventricle, etc., and which in adequate doses removes the symptoms of diabetes for several hours. The extract must be given subcutaneously twice daily. Opium in crude form or its alkaloid codein promotes comfort by obtunding perceptions and may diminish slightly the glycosuria by retarding the absorption of carbohydrates, but its use is inadvisable except in hopeless cases. Tonics are sometimes indicated. Constipation, which is baneful, if not relieved by food substitutes (bran, agar agar, etc.) must be combated by vegetable or saline cathartics. Courses of bromids are sometimes of service in controlling nervous manifestations.

Most of the complications of diabetes are controlled or greatly benefited by strict dieting. Boils usually yield to vigorous antidiabetic treatment. Pruritus of the genitals may be relieved by anointing the parts freely with petrolatum before urination or protecting them with zinc stearate. General pruritus may require the use of a wash containing phenol, resorcinol or boric acid.

Diabetic Coma.—If coma is impending and the patient has been on an ordinary diet, a moderate amount of carbohydrate in the form of thin oatmeal gruel made with water (60 grams of oatmeal each twenty-four hours for a patient weighing 150 pounds) should be ordered. Sodium bicarbonate seems to be helpful in replenishing the alkali reserve, although Joslin believes that it does more harm than good. It may be given by the mouth (4 to 6 gm. or more in weak solution) every two hours or by the rectum (3 to 5 per cent. solution). In urgent cases, however, it is best given intravenously (not subcutaneously) in 4 per cent. solution made with freshly sterilized water. For an adult 500 mls or more may be injected every few hours. A neutral reaction of the urine or, better, a normal blood CO₂ reading, is an indication that sufficient alkali has been given. Solutions of sodium bicarbonate should not be boiled as the heat tends to transform the bicarbonate into carbonate, which is injurious. Whether alkali is used or not, a large amount of fluid (1000 mls within each six hours) should be prescribed. The fluid may be taken by the mouth as water, tea, coffee or thin broths, or by the rectum or intravenously in the form of normal saline solution. Nausea must be avoided and every effort that excites it should be suspended. Free evacuation of the bowels is necessary and may be secured by calomel and salts or by enema. Recently, remarkable effects have been reported from the use of insulin in diabetic coma.

DIABETES INSIPIDUS

Definition.—A chronic condition, characterized by the excretion of large quantities of pale, limpid urine of low specific gravity and free from albumin and sugar.

Etiology.—The disease is most common between the ages of twenty and thirty. More males are affected than females. Heredity may be an important factor. Heredity is occasionally a factor. In the majority of cases diabetes insipidus is caused by a lesion of hypophysis (especially the posterior lobe) or an adjacent structure at the base of the brain. Frequently

the lesion is of syphilitic origin. In other cases the kidneys seem to be incapable of secreting a concentrated urine, that is, urine containing a normal percentage of salt and urea.

Symptoms.—The onset may be gradual or sudden. The chief symptom is the passage of large quantities (5 to 20 liters) of sugar-free urine, of low specific gravity (1005–1001), over a long period. Accompanying the polyuria there is usually insatiable thirst. The skin and mouth are dry; the bowels are usually constipated, and headache, lumbar pains and nervous irritability are common. In many cases there is neither weakness nor emaciation. Complications are rare.

Diagnosis.—The high specific gravity of the urine and the presence of sugar will serve to distinguish *diabetes mellitus* from diabetes insipidus.

Chronic nephritis may be recognized by the presence of tube-casts in the urine, the albuminuria, and cardiovascular signs.

Prognosis.—The disease usually continues for years and is not often directly fatal. Complete cure is rarely effected by treatment, even in syphilitic cases, but it occasionally occurs spontaneously.

Treatment.—It is not advisable to restrain the patient much in the matter of drink, except in the evening when the intake of fluid should be reduced, so as to avoid interference with sleep. A salt-poor diet is sometimes beneficial. Arsphenamin, mercury and iodids should be given a thorough trial whenever there is evidence of syphilis. Subcutaneous injections of an extract of the posterior lobe of the pituitary body (1 mil once a day) often give great relief, although the action of the drug rarely lasts more than twenty-four hours. Oral administration is ineffectual. In a few cases lumbar puncture has been followed by marked improvement or actual cure. Among special remedies that have been extolled from time to time may be mentioned valerian ($1\frac{1}{2}$ fluidounce of the ammoniated tincture daily), ergot (10 minims of the fluidextract three times a day), strychnin sulphate ($1\frac{1}{30}$ – $1\frac{1}{20}$

grain two or three times a day hypodermically), and the bromids.

SCURVY

(Scurbutus)

Definition.—Scurvy is a disorder of nutrition due to defective diet and characterized by weakness, anemia, swollen spongy gums, and a tendency to hemorrhages.

Etiology.—In adults the chief etiologic factor is a lack of fresh food, especially of succulent vegetables and fruits. Unhygienic surroundings, exposure to cold and wet, and mental depression are cited as predisposing causes. The essential element which is lacking in the food is apparently a vitamin, probably the water-soluble vitamin C.

Symptoms.—These include anemia with great weakness and lassitude; spongy, bleeding gums with fetor of the breath and loosening of the teeth; subcutaneous ecchymoses and hemorrhages from the mucous membranes; and brawny induration of the muscles in various parts of the body from a sanguineous transudation.

An *infantile form of scurvy* (Barlow's disease) is not uncommon. It usually follows the exclusive use of condensed milk, sterilized milk, or proprietary foods, and develops, as a rule, between the ages of 6 and 18 months. The characteristic symptoms are: Pallor, tenderness or pain in the legs or back on handling, slight swelling, especially about the diaphyses, immobility of the legs (pseudoparalysis), ecchymoses, and hematuria. The gums are not affected unless the child has teeth. The disease is often confused with rheumatism, but the latter is rare in children under 2 years of age, produces swelling in the joints rather than the bony shafts, and is more likely to be accompanied by fever and local heat than scurvy.

Prognosis.—Recovery usually occurs rapidly under appropriate treatment, but neglected cases may be fatal.

Treatment.—The diet should include fresh vegetables—potatoes, lettuce, cabbage, and onions—with several ounces

of lemon-juice daily. Iron is of service. The mouth should be cleansed at frequent intervals with an antiseptic wash.

In infantile scurvy good results follow the use of raw milk, orange-juice (1 tablespoonful daily), and purée of potato.

BERIBERI

(Endemic Multiple Neuritis; Kakke)

Definition.—A form of polyneuritis occurring endemically in tropical and subtropical countries and characterized by disturbances of sensation, of motion, and of the circulation.

Etiology.—Beriberi is especially prevalent in Japan, China, the Philippine Islands, India, and parts of South America. Although the etiology of the disease has not been definitely determined, the experiments of Fraser and Stanton, confirmed by other workers, are conclusive to the effect that a defective diet is an essential factor. Outbreaks have usually been traceable to the too exclusive feeding on highly polished rice or other carbohydrate food which is deficient in an antineuritic vitamin (water-soluble B). Overcrowding, bad hygienic surroundings, and exposure to wet are predisposing factors. Young adults are chiefly affected.

Symptoms.—To the usual symptoms of polyneuritis (paresis, especially in the lower extremities, pains, paresthesia, hypesthesia, tenderness of the nerve-trunks, loss of deep reflexes, diminished electric excitability, and muscular atrophy) are added various disturbances of the circulation, such as palpitation, dyspnea, weakness of the pulse, and signs of venous congestion. In many cases more or less edema of the feet and legs is also noted.

Varieties.—*Acute pernicious* forms occur in which the onset is rapid, grave disturbances of the circulation soon develop, and death ensues in the course of a few days from heart failure and edema of the lungs. So-called *wet* and *dry* forms are also observed. In the former there is marked edema and often effusions into the serous sacs. In the latter there is little or no

edema, but muscular atrophy is very pronounced. Finally, in all outbreaks there are many *rudimentary* cases in which the only symptoms are weakness in the legs, paresthesia, and palpitation.

Prognosis.—The mortality has varied in different outbreaks from 2 to 60 per cent. Generally speaking, the prognosis is good, except in pernicious cases, when the patient can be placed under proper hygienic conditions. The average duration of cases of moderate severity is between three and eight weeks.

Treatment.—Good hygienic conditions and a liberal diet containing especially unpolished rice, whole wheat, barley, yellow corn-meal, fresh vegetables and fruits are the most important prophylactic measures. A person sick with beriberi should be confined to bed and given foods rich in antineuritic vitamin—raw milk, eggs, rare beef juice, barley soup, extract of rice polishings and malt extract. Saline laxatives appear to be useful in the early stages. Morphin is serviceable when there is much pain or dyspnea. Caffein and the vegetable salts of potassium are worthy of trial if there is marked edema. Venous congestion is sometimes favorably influenced by venesection. For cardiac dilatation digitalis strophanthin (intramuscularly) and caffein are the best drugs. In the chronic stages the treatment is that laid down for multiple neuritis (see p. 520).

PELLAGRA

Definition.—Pellagra is an endemic chronic disease characterized by erythematous roughness of the skin, digestive disturbances, and nervous symptoms, with seasonal exacerbations and remissions, and a tendency to terminate in dementia and exhaustion.

Etiology.—The disease prevails especially in southern Europe, the West Indian Islands, and in the southern parts of the United States. The cause is obscure. According to one theory pellagra is in some way associated with the continuous

use of spoiled maize, and according to another theory it is a result of the lack of some essential dietary substance or vitamin. The weight of evidence seems to favor the view that it is caused by a lack of proper food balance.

Symptoms.—The digestive disturbances are usually the first symptoms, and comprise stomatitis and glossitis, abdominal pains, and recurring attacks of diarrhea. Vomiting is not uncommon. The cutaneous eruption appears in the spring and disappears or recedes in the winter. It is most frequently found on the dorsal aspect of the hands and feet, the neck and the face, and takes the form of a desquamative erythema or dermatitis. The nervous symptoms consist of neurasthenia, mental depression, paresthesias, and muscular cramps, and in the later stages, hallucinations, delusions of a persecutory character, and dementia.

Prognosis.—The outlook in the early stages is good, but it is unfavorable when the disease well developed. The average duration is probably about 5 years.

Treatment.—Improvement in the environment and food supply of the people is the chief element in prophylaxis. In the treatment a varied nutritious diet, removal to suitable hygienic surroundings, rest, and hydrotherapy are all important. Arsenic in the form of Fowler's solution, sodium cacodylate or arsphenamin is sometimes of service. A combination of nux vomica and hydrochloric acid is also of value as an aid to digestion.

DISEASES OF THE NERVOUS SYSTEM

DISTURBANCES OF MOTILITY

These consist for the most part of loss of motor power or paralysis (akinesis) and various symptoms of exaggerated motility (hyperkinesis), such as convulsions, choreic movements and tremors.

Motor Paralysis.—Paralysis affecting one member is known as *monoplegia*; affecting a lateral half of the body, as *hemiplegia*; and affecting the two lower limbs, as *paraplegia*. Partial loss of motor power is termed *paresis*.

Monoplegia may result from:

1. A focal lesion in the cortical area of the brain. This may be recognized by the history, increased tonicity of the muscles, exaggerated deep reflexes, and absence of muscular atrophy and of degenerative electrical changes.

2. A lesion of the peripheral nerves. This may be recognized by the history, loss of deep reflexes, loss of muscular tone and later, atrophy, degenerative electrical changes, and usually the presence of sensory disturbances.

3. Hysteria. This may be recognized by the history, the emotional instability and pathologic suggestibility of the patient, and the absence of wasting, alterations in tendon reflexes, and all other signs of organic disease.

Hemiplegia may result from:

1. A diffuse lesion of the motor cortex. The paralysis is on the opposite side of the body and is unassociated with anesthesia.

2. A lesion of the internal capsule. This is the most common seat of hemorrhage; the paralysis is on the opposite side of the body and is rarely associated with anesthesia.

3. A lesion of a cerebral peduncle, this frequently gives rise to hemiplegia on the opposite side, and oculomotor palsy (dilatation of pupil, ptosis and strabismus) on the side of the lesion.

4. A lesion of the pons. This frequently produces hemiplegia and hemianesthesia on the opposite side, and palsy of one or more cranial nerves, especially the facial, trigeminus or abducens, on the side of the lesion.

5. A unilateral lesion high in the cord (very rare). This produces on the *affected side* atrophic paralysis, with anesthesia at the level of the lesion, and spastic paralysis with loss of muscular sense below the lesion, and on the *opposite side* anesthesia below the level of the lesion ("Brown-Séquard's paralysis").

6. Hysteria. This presents the same characteristics as hysteric monoplegia.

Paraplegia may result from:

1. Acute myelitis. This develops in the course of a few days, usually as the result of trauma, syphilis, or an acute infection; the paralysis is flaccid at first, but gradually becomes spastic; sphincter disorders, anesthesia, and frequently a "girdle sense" are present, and there is a marked tendency to bed-sores and cystitis.

2. Landry's disease (acute ascending paralysis). This resembles myelitis in mode of onset, but the paralysis is unassociated with sphincter disorders, anesthesia, bed-sores, or other trophic disturbances.

3. Acute poliomyelitis. This occurs chiefly in children; it comes on suddenly, and is ushered in with fever, and, frequently, with pains in the limbs; the palsy develops very rapidly, affects groups of muscles rather than entire members, is flaccid and atrophic, and tends toward recovery in some parts; sphincteric incontinence and anesthesia are absent, and there is no tendency to bed-sores.

4. Compression of the cord (spinal caries, tumor, aortic aneurysm, etc.). The suggestive features are localized pain, tenderness, rigidity, and deformity; stabbing pains in the limbs; parasthesia, followed by anesthesia; progressive spastic paralysis, and interference with the sphincters. Roentgen examination is of much value in diagnosis.

5. Hemorrhage into the spinal cord or spinal membranes. The history of trauma or strain is important. In the former the paralysis is abrupt, and pain, if present, is usually localized. In the latter, pain is intense and diffuse, while paralysis is a subordinate feature.

6. Fracture or dislocation of the spine. The history of injury, the local deformity, the roentgen findings, the abrupt onset of flaccid paraplegia, with anesthesia and sphincteric interference, and the marked tendency to bed-sores and cystitis are the diagnostic features.

7. Some forms of multiple neuritis. The history of some antecedent infection or intoxication, diffuse pain, tenderness of the nerve-trunks and calves, the special incidence of the palsy and sensory disturbances on the peripheral parts of the limbs, muscular atrophy, and loss of tendon reflexes, without sphincter disorder, girdle sensation, or any marked tendency to bed-sores, will point to neuritis.

8. Amyotrophic lateral sclerosis and lateral sclerosis. Both of these conditions develop gradually, and are marked by motor weakness, spasticity of the muscles, and exaggerated deep reflexes, without sensory symptoms. In the former there is also muscular atrophy.

9. Disseminated sclerosis. In this there is spastic paresis in association with intention tremor, scanning speech, nystagmus, and visual defects.

10. Cerebral palsies of childhood. These are characterized by spastic paralysis, mental deficiency, stunted physical development, and, also, in many cases, by choreiform movements and epileptic seizures.

11. Caisson disease (divers' paralysis). The history will establish the diagnosis.

12. Hysteria. This may be recognized by the emotional instability and suggestibility of the patient, and by the absence of wasting, sphincteric disturbances, alterations in deep reflexes, and all other signs or organic disease.

Convulsions.—Convulsions are involuntary muscular contractions, interrupted or long-continued, involving a large part of the skeletal system, and resulting from excessive irritation or undue irritability of the motor centers.

Interrupted contractions occurring in rapid succession are termed *clonic* and long-continued contractions are termed *tonic*.

Convulsions may be general or local. The term *spasm* is sometimes applied to the latter.

General clonic convulsions may be due to:

1. Essential epilepsy. This condition usually develops before the age of twenty-five, and the convulsions are general and are unassociated with any definite cause.

2. Organic brain disease. In this condition there may be a history of syphilis or of injury; the convulsions may be local, or begin as such and become general; and there may be concomitant symptoms of cerebral disease.

3. Various intoxications. Alcoholism, the infectious fevers, and uremia are frequently associated with clonic convulsions.

4. Reflex irritation. Such convulsions are usually observed in infants, and result from gastric irritation, an adherent prepuce, intestinal parasites, or teething.

5. Cerebral anemia. Such convulsions are sometimes seen after profuse hemorrhage, and in degeneration of the heart muscle (Adams-Stokes syndrome).

Tonic convulsions may result from:

1. Tetanus. This may be recognized by the presence of a wound, the early involvement of the muscles of mastication, and the persistent rigidity of the muscles of the back.

2. Cerebrospinal meningitis. This may be recognized by the headache, fever, rigidity of the neck, Kernig's sign, local palsies, and characteristic changes in the spinal fluid.

3. Strychnin-poisoning. This may be recognized by the history, the sudden onset, the intermittent character of the tonic contractions, the absence of fever, and the escape of the muscles of the jaw until very late.

4. Tetany. In this condition the extremities are chiefly involved; the contractions are tonic and intermittent, involve bilaterally symmetrical groups of muscles, and can be reproduced by pressure on the nerves of the affected member.

Hysteric Convulsions.—The convulsions of hysteria may simulate those of epilepsy, but, unlike the latter, they are rarely attended by complete loss of consciousness, pupillary rigidity, biting the tongue, or involuntary passage of urine. Moreover, in hysteria the movements are usually tonic, or if clonic, appear purposive, and the paroxysms are of long duration.

Eclampsia.—This term is applied to a sudden attack of convulsions, the result of a temporary cause, such as the convulsions of childhood resulting from reflex irritation, and the convulsions of pregnancy resulting from toxic substances retained in the blood.

Local Convulsions or Spasms.—*Facial spasm* may result from—(1) Irritative lesions at any point in the course of the seventh nerve from the cortex to the periphery; (2) peripheral facial palsy (Bell's palsy); (3) tic, in which there are natural but purposeless and inopportune contractions of psychogenic origin; (4) neuralgia of the sensory division of the seventh nerve (geniculate neuralgia).

Transitory spasms of one arm or one leg are usually manifestations of Jacksonian epilepsy (focal epilepsy), but they sometimes result from hysteria.

Spasm of the hand developing when the member is put to use may result from writers' cramp, myotonia congenita (Thomsen's disease), or hysteria.

Spasm of the cervical muscles (wry-neck, torticollis) may be due to: (1) Exposure to cold (acute "stiffneck"); (2) congenital shortening of sternomastoid muscle; (3) irritation of the spinal accessory nerve caused by disease of the vertebræ or meninges in the upper cervical region; (4) obscure functional disturbance of the cortical centers (mental torticollis, psychogenous tic).

Spasms of the larynx, esophagus, and diaphragm (hiccup) have already been discussed.

Choreiform Movements.—These are involuntary, jerky, quickly changing muscular contractions resulting in movements that are of a purposeless nature, but not wholly incoördinate. Chorea must not be confused with *convulsive tic*, in which the movements are purposive and coördinate, and unnatural only in their involuntary reproduction, inopportuneness, and frequency of repetition. Choreic movements may result from:

1. Infectious chorea (St. Vitus' dance). This disease occurs chiefly in children, usually lasts from six to ten weeks, is prone to recur, and is frequently complicated by endocarditis. A severe form occurring chiefly in women during pregnancy and characterized by violent movements, fever and delirium, is known as chorea insaniens.

2. Huntingdon's chorea. This affection occurs in adult life and is hereditary. The movements in time become general involving the muscles of speech and deglutition, and are associated with a progressive mental deterioration.

3. Cerebral diplegia and hemiplegia. Choreiform movements are frequently observed in the cerebral paralysis of children and occasionally they occur in adults on the paralyzed side after cerebral apoplexy (post-hemiplegic chorea).

4. Senile chorea. Occasionally old persons with arteriosclerosis and degenerative changes in the brain become subject to chorea.

5. Friedreich's ataxia. Choreic movements are not uncommon in this disease, which may be recognized by the ataxic gait, nystagmus, disturbances of speech, and family history.

6. Epidemic Encephalitis—choreiform movements are among the hyperkinetic disturbances frequently observed in epidemic encephalitis.

Athetosis.—This term was suggested by Hammond to designate certain movements occurring chiefly in the hands and feet, and characterized by slow twisting, intertwining, separation, and extension of the fingers and toes. Athetosis is virtually a form of chorea. It is observed most frequently in infantile cerebral paralysis.

Tremors.—A tremor is a rhythmic vibratory movement produced by the alternate contraction and relaxation of antagonistic muscles. The tremor is said to be fine when the oscillations are 8 to 12 per second, and coarse when the oscillations are 4 to 8 per second. An *intention tremor* is one that only becomes distinct when the patient attempts purposeful movements. Tremors are observed in (1) health after excitement or excessive physical exertion; (2) in the convalescence from exhausting disease; (3) senility; (4) certain neuroses, especially neurasthenia and hysteria; (5) certain intoxications, as from alcohol, morphin, mercury etc.; (6) certain organic diseases of the central nervous system, notably, disseminated sclerosis, progressive lenticular degeneration, and parietic dementia; (7) paralysis agitans; hyperthyroidism.

Myoclonia is a term used to designate brief clonic contractions of various sorts, such as choreic movements, tics, and fibrillary twitchings or wave-like contractions of individual muscles producing no locomotor effect. To the last the term *myokymia* is usually applied. Myokymia is observed especially in muscles undergoing atrophy from neurotrophic influence (muscular dystrophies, bulbar palsy, etc.). It may occur also without atrophy or paresis in epidemic encephalitis. Closely related to it are the rapid wave-like contractions involving the whole muscle occurring in the *myoclonus multiplex of Friedreich*. In this rare disease, which is probably of infectious origin, rapid wave-like contractions, at a rate of

from 20 to 50 a minute, occur bilaterally in the individual muscles of the arms, thighs, trunk and face.

The Gait.—*The Ataxic Gait.*—In locomotor ataxia the patient raises the feet abnormally high, throws them out loosely, and brings them down abruptly with a stamp.

Spastic Gait.—In spastic paraplegia the movements are stiff, the knees come together, the leg drags behind, and the toe catches the ground.

Festination.—This term is applied to the gait of advanced paralysis agitans. In walking, the body inclines more and more forward, and the steps grow faster and faster until the patient falls, straightens himself by a supreme effort, or grasps some support.

Steppage Gait.—In chronic multiple neuritis the patient raises the foot high, turns the toe up, and brings the heel down first.

The Gait of Pseudomuscular Hypertrophy.—The feet are wide apart, the belly protrudes, and the movements are clumsy and waddling.

Titubation.—This term is applied to the peculiar gait observed in lesions of the cerebellum. It resembles the gait of locomotor ataxia, but is much more staggering, the body swaying like that of a person intoxicated. With the ataxia there is a marked vertigo, which usually disappears when the patient lies down.

The Reflexes.—*The Knee-jerk, or Patellar Tendon Reflex.* This is obtained by tapping the quadriceps tendon between its insertion and the patella while the leg is crossed over its fellow.

The knee-jerk is increased in the following conditions:

1. In cerebral lesions involving the pyramidal tracts (diminution of inhibition), as in hemiplegia from apoplexy, tumor, etc.

2. In compression or partial destruction of the cord above the lumbar region, as in Pott's disease or tumor of the cord.

3. In disseminated cerebrospinal sclerosis, lateral sclerosis, and amyotrophic lateral sclerosis.

4. In irritability of the cord, as in mania, hysteria, strychnin-poisoning, and spinal meningitis.

The knee-jerk is diminished or absent in the following conditions:

1. In the various forms of muscular dystrophy.
2. In lesions of the nerves which cut off the impulse from the cord—as neuritis.
3. In lesions of the posterior columns of the cord, as in locomotor ataxia.
4. In poliomyelitis, both acute and chronic.
5. In transverse lesions of the cord completely destroying the reflex arc, as in lumbar myelitis, fracture of the vertebræ, etc.
6. In exhaustion of the spinal centers, as after prolonged muscular exertion.

Ankle-clonus.—This consists of vibratory movements of the foot, obtained by supporting the *tendo Achillis* with one hand while the foot is strongly flexed with the other. It occurs with lesions that incompletely interrupt the conductivity of the pyramidal tracts, such as primary lateral sclerosis, amyotrophic lateral sclerosis, disseminated sclerosis, degeneration of the lateral columns following cerebral hemorrhage, and compression myelitis.

Arm-jerk.—This is obtained by striking the biceps tendon at the elbow, or the triceps tendon above the olecranon.

Jaw-jerk.—This is obtained by tapping the jaw while the mouth is partially open.

Kernig's Sign.—This consists in an inability to straighten the leg completely when the patient is in the recumbent posture and the thigh is flexed at a right angle with the pelvis. It is of value in the diagnosis of meningitis.

Cutaneous or Superficial Reflexes.—These are muscular contractions resulting from irritation of the sensory nerves of the skin. Only three are especially important.

Babinski's Reflex.—This consists in extension of the great, sometimes with a fan-like spreading out of the other toes,

when the sole of the foot, especially along the inner side is stroked with a blunt pencil. It is often normally present in infants. In adults it is almost pathognomonic of disease of the pyramidal tracts.

Cremasteric Reflex.—This consists in elevation of the testicle (not merely of the scrotum) upon stroking or scratching the inner side of the thigh.

Abdominal Reflex.—This consists in sudden contraction of the abdominal muscles upon stroking the skin on one side of the abdomen.

The presence of the cremasteric and abdominal reflexes indicates that the spinal cord at the level through which the impulses pass is healthy. Absence of these reflexes, however, is not especially significant.

DISTURBANCES OF SENSATION

These consist chiefly in a loss of sensibility—*anesthesia*; increased sensibility—*hyperesthesia*; subjective sensations, not actually painful—*paresthesia*; and subjective painful sensations—*neuralgias*.

Anesthesia.—Ordinary cutaneous sensibility may be tested by the prick of a pin, by a pinch, or by the faradic current. Diminution of sensibility is known as *hypesthesia*.

In cerebral lesions involving the upper sensory neurons anesthesia is usually incomplete and associated with hemi-anesthesia. In lesions involving individual segments and roots of the spinal cord the anesthesia occurs in bands, known as segmental or radicular zones. Thus, when a single dorsal segment is destroyed there is anesthesia in the sensory area represented in that segment and also in the areas represented in all the segments below the first; but when the nerve roots alone are destroyed the anesthesia is confined to the area represented by the particular segments with which injured nerve roots correspond (root type of anesthesia). In lesions of peripheral nerves the anesthesia occurs in areas of the skin supplied by the affected nerves. In psychic disturbances

affecting sensation (hysteria) the areas of anesthesia do not correspond to the distribution of any particular nerves, roots or segments.

Analgesia and Thermo-anesthesia.—The former is a loss of sensibility to pain, the latter a loss of sensibility to heat and cold. The association of these two disturbances, together with the preservation of tactile sensation, is known as *dissociative anesthesia*. This phenomenon is most frequently observed in syringomyelia, but it may occur also, though rarely, in spinal tumor, vertebral caries, locomotor ataxia, neuritis, and hysteria.

Impairment of Deep Sensibility or Bathyanesthesia.—This may involve the sense of position of movement, of resistance and weight, or of vibration. Bathyanesthesia occurs when the impulses (proprioceptive) from the muscles, joints and tendons fail to reach the brain owing to a lesion of the peripheral nerves or conducting tracts in the spinal cord (columns of Goll and Burdach) or when a cortical lesion exists which interferes with the perception and interpretation of such impulses.

Pallanesthesia, or the loss of the peculiar vibrating or trembling sensation which is normally felt when the foot of a large tuning fork is firmly placed in contact with a bony prominence, such as the malleoli, the styloid process of the ulna or the sternum, is frequently one of the earliest indications of multiple neuritis, tabes dorsalis, combined posterolateral spinal sclerosis, and compression myelitis. It is not observed in function conditions, such as hysteria.

Astereognosis.—This is an inability to recognize objects by handling them. While it is present when there is complete tactile anesthesia, it also occurs when the tactile sense is intact if deep sensibility is lost as the result of a peripheral lesion, or if the cerebral center for deep sensibility or for the perception of form is injured. Central astereognosis without complete tactile anesthesia is usually a result of a lesion in the parietal lobe.

Ataxia of motion is impairment of the coördination necessary to the accomplishment of purposeful movements. It depends upon a disturbance of the deep sensibility of the extremities or an inability of the centers to carry out normally coördinated movements. It is observed typically in tabes dorsalis, Friedreich's disease, polyneuritis, and lesions of the motor cortex (cortical ataxia).

Lesions of the cerebellum cause a form of incoördination known as asynergy, which depends upon a disturbance of the mechanism of equilibration and of that which enables one to move the segments of the body in proper degree and rhythm (Dana). The gait is reeling like that of a drunken person and is quite different from that of the tabetic.

Hyperesthesia is excessive sensibility to sensory stimuli. It may be produced by irritation of any portion of the sensory tract.

Paresthesia.—This term is used to indicate certain disagreeable subjective phenomena, such as numbness, tingling, itching, creeping, prickling, etc.

Paresthesia is observed in various conditions causing slight irritation of the sensory tract, such as neuritis, tabes dorsalis, and hysteria. It is frequently observed in elderly persons with general arteriosclerosis.

Girdle Sensation.—The sensation of having a girdle or tight band around the trunk. It is frequently observed in locomotor ataxia and myelitis.

Neuralgia.—This consists of paroxysms of severe pain radiating along the line of the nerve-trunks. The pain is relieved by pressure, but there are tender spots (*points douloureux*) where the nerve makes its exit from bony canals or muscular coverings.

Lightning-pains.—This term is applied to the sharp lancinating pains observed in locomotor ataxia. They usually occur in the extremities, and are sometimes ascribed to rheumatism.

DISTURBANCES OF NUTRITION

These consist chiefly of atrophy of the muscles, changes in the joints, and alteration in the skin and its appendages.

Muscular Atrophy.—Atrophy or wasting of the muscles may result from:

1. Inactivity. Cerebral palsies may thus be associated with gradual wasting (simple atrophy).

2. Certain affections of the muscles themselves, as the various types of progressive muscular dystrophy (simple atrophy).

3. Diseases affecting the anterior horns of gray matter of the spinal cord or the motor nerves (degenerative atrophy).

Changes in Electromuscular Contractility.—A normal response of the muscles to both galvanic and faradic currents usually occurs in *hysteric paralysis* and in *paralysis of cerebral origin*. An increased response to both currents without qualitative change indicates a state of hypersensitiveness of the spinal centers or peripheral nerves, and may be observed in *very recent cases of neuritic paralysis* and in *tetany*. A diminished response to both currents without qualitative change is frequently observed in the *muscular dystrophies*.

Reaction of Degeneration (DeR).—This consists in a qualitative change in the electric reaction, a reversal of that occurring in normal muscle. It is obtained only with the *galvanic current* when the electrode is placed over the *muscle*—not its motor nerve or motor point—and occurs in paralyzed muscles which are in certain stages of degeneration owing to a lesion of the ganglion cells in the anterior gray horns of the cord or of the prolongations of these cells in the peripheral nerves. Thus it is observed in *acute* and *chronic poliomyelitis*, in *acute myelitis*, and in *severe forms of neuritis*. In these diseases the affected muscles fail to respond to the faradic current, but still respond to the galvanic current. The responses, however, instead of being prompt and short, as in health, are sluggish and persistent, and, moreover, are reversed

in their sequence. Thus, the anodal (positive pole) closing contraction may equal, or at a later period exceed, the cathodal (negative pole) closing contraction, and the cathodal opening contraction may equal or exceed the anodal opening contraction. These reactions may be expressed as follows:

AnCIC equals or is greater than CaCIC. CaOC equals or is greater than AnOC.

Arthropathies.—An arthropathy is a degenerative affection of the joints, characterized by marked swelling due to effusion, erosion of the cartilages, relaxation and calcification of the ligaments, and atrophy of the heads of the bones. Arthropathies are observed in certain organic diseases of the spinal cord, more especially in locomotor ataxia and syringomyelia.

Ulceration Resulting from Perverted Nutrition.—*Acute Decubitus.*—This term is applied to ulcers appearing in a few hours or days, on parts subjected to pressure, after the occurrence of a severe cerebral or spinal lesion.

Chronic Decubitus.—This term is applied to the ulcers which ultimately appear on parts subjected to pressure in the course of chronic spinal affections.

Perforating Ulcer of the Foot.—This term is applied to an undermining ulcer of the foot most commonly observed in locomotor ataxia. It frequently penetrates the deep structures and involves the bones.

Symmetric Gangrene (Raynaud's Disease).—This is a gangrenous affection involving the fingers, toes, tip of the nose, or ears. It arises spontaneously, and is probably due to a vasomotor spasm.

Trophic Affections of the Skin.—Herpes, scleroderma, vitiligo, chloasma, and "glossy skin" following injuries of the nerve-trunks are illustrations of this class of trophic phenomena.

Trophic Affections of the Hair and Nails.—After injury of the nerves and in neuritis the nails often become dry, brittle, and cracked. Under similar conditions there may be a loss of hair, an overgrowth of hair, or a change in the color of the hair.

DISTURBANCES OF CONSCIOUSNESS

Coma.—Coma is a state of prolonged unconsciousness, somewhat resembling sleep, from which the patient cannot be aroused.

Temporary unconsciousness due to anemia of the brain is termed *syncope*. It may be recognized by the extreme pallor, weak pulse, and feeble heart-sounds. Coma may result from:

1. *Traumatism*.—This can be recognized only by the history or the local evidence of injury (bleeding from the ear or nose, ecchymosis about the eye or mastoid process, etc.).

2. *Organic Disease of the Brain*.—The most common cause under this head is apoplexy, which may usually be recognized by the history, the age, the condition of the arteries, and by evidences of paralysis, such as stertorous breathing, unnatural relaxation or rigidity on one side of the body, conjugate deviation of the eyes, and drooping of one corner of the mouth.

3. *Epilepsy*.—The coma of epilepsy is usually of short duration. It may be recognized by the history, by the bloody saliva, by the presence of scars on the tongue from previous attacks, and by the exclusion of other causes.

4. *Thermic Fever (Sunstroke)*.—The temperature of the day or of the room in which the patient is found, the extremely high body-temperature, and the absence of symptoms of other conditions causing coma will usually prevent an error in diagnosis.

5. *Certain Drugs*.—Acute alcoholism and acute opium poisoning are familiar examples. In *alcoholism* the patient can usually be aroused by shouting in the ear, there is the characteristic odor on the breath, and other causes can usually be excluded.

In *opium-poisoning* the pupils are small, the respirations are very slow, the temperature is normal or subnormal and there are no signs of paralysis.

6. *Uremia*.—Coma from this cause is often interrupted by convulsions; the aortic second sound is often accentuated; the

urine contains albumin and casts; the temperature may be above or below normal; the pupils are usually small and equal, albuminuric retinitis may be present; and examination of the blood may show an excess of waste nitrogen.

7. *The Infectious Fevers*.—The diagnosis is usually suggested by the clinical history. Pernicious malarial fever may produce sudden coma, and in this condition the examination of the blood affords conclusive evidence.

8. *Hysteria*.—The coma has the appearance of normal sleep; the patient can usually be aroused by painful stimuli, as pressure over the supraorbital nerve, or the suggestion of some painful procedure; the temperature, pulse and respiration are not usually altered, and there is often a history of other hysterical manifestations.

9. *Diabetic Acidosis*.—Diabetic coma may be recognized by the history, the fruity odor of the breath, the glycosuria, and the presence of acetone bodies in the urine.

Catalepsy.—This is a state of mental and motor inertia in which the limbs tend to remain for long periods in any position in which they are placed. During the attacks the patient is apparently insensible to external impressions. It occurs in hysteria, hypnosis, and certain psychoses, especially dementia præcox.

DISTURBANCES OF THE SPECIAL SENSES

The Eye.—*Myosis*.—Contraction of the pupil occurs in many conditions, notably in locomotor ataxia, paretic dementia, syringomyelia, early stages of increased intracranial pressure, paralysis of the cervical sympathetic, uremia, opium-poisoning, and old age.

Mydriasis.—Dilatation of the pupil is also observed in many conditions, notably in atrophy of the optic nerve, paralysis of the third nerve, severe pain, epileptic seizures, hysteric attacks, belladonna-poisoning, and in asphyxia from any cause.

Inequality of the Pupils.—Slight inequality may occur in health. Marked inequality (*anisocoria*) may be seen in ocular defects, in organic brain disease, in parietic d ementia, in locomotor ataxia, in aneurysm pressing on the cervical sympathetic, and in unilateral paralysis of the oculomotor nerve.

Argyll-Robertson Pupil.—This is one that reacts to distance but not to light. It is noted especially in locomotor ataxia and parietic dementia.

Conjugate Deviation of the Eyes.—This term is applied to the forcible deflection of the eyes to one side, the visual axes still remaining parallel. It is a common symptom in gross lesions, such as hemorrhage or tumor, of the motor centers or their tracts in the brain. When the lesion is cerebral and destructive the eyes are turned away from the palsied side (toward the lesion) and when the lesion is cerebral and irritative the eyes are turned toward the convulsed side (away from the lesion). In pontine lesions the deviations are exactly reversed.

Nystagmus.—This is tremor or rapid oscillation of the eyeball. It may be congenital; it occurs with various defects of vision; it is not uncommon in miners (muscle fatigue); it occurs in labyrinthine disease; it is observed in certain organic diseases of the brain, especially disseminated sclerosis, Friedreich's ataxia and tumors of the cerebellum.

Optic Neuritis or Papillitis.—An inflammatory affection of the intra-ocular end of the optic nerve. The term "choked disk" is used to designate the condition when it is accompanied with marked swelling. Its chief causes are: Tumor of the brain, cerebral meningitis, syphilis, toxic agents (lead and alcohol), infectious fevers, anemia, and renal disease.

Atrophy of the Optic Nerve.—As a primary affection it is most commonly observed in locomotor ataxia and parietic dementia. Secondary atrophy results from pressure of tumors, aneurysms, etc., on the optic chiasm. Consecutive atrophy is a sequel of optic neuritis.

The Ear.—*Tinnitus Aurium (Subjective Noises in the Ear).*—This is observed in cerebral hyperemia and anemia, in dis-

eases of the ear, in Ménière's disease, and after the use of certain drugs, as quinin and salicylic acid.

Hyperacusis of Hearing.—This is sometimes observed in hysteria, in facial paralysis, and in cerebral hyperemia.

Deafness usually depends upon disease of the ear itself.

PSYCHIC DISTURBANCES

Delusion.—A delusion is a faulty belief concerning a subject capable of physical demonstration, out of which the person cannot be reasoned by adequate methods for the time being (Wood).

A *systematized delusion* is one which the patient endeavors to defend by a process of reasoning more or less logical. Systematized delusions are especially observed in paranoia.

An *unsystematized delusion* is one which the patient makes no attempt to justify; he asserts his belief without reason. The majority of delusions are unsystematized, and as such are observed in many psychoses.

A *fixed delusion* is one which the patient retains for a considerable length of time; it is frequently systematized. Fixed delusions are observed in paranoia and sometimes in parietic dementia and melancholia.

An *expansive delusion* or a *delusion of grandeur* is one which exalts its possessor. The patient conceives that he is some noted personage, that he is worth millions of dollars, or that he is capable of performing certain marvelous feats. Expansive delusions are frequently observed in parietic dementia, mania, and paranoia.

A *hypochondriacal delusion* is one which depresses its possessor. Thus, the patient may believe that he has committed the unpardonable sin, that he is being persecuted, or that he is the victim of some dread disease. Hypochondriacal delusions are frequently observed in melancholia, alcoholic insanity, and in some cases of paranoia and parietic dementia.

Illusion.—An illusion is a perverted perception. Thus, the patient sees an object in his room and perceives it as a demon,

or he hears the clock-tick and imagines it to be a voice. Illusions are observed in various psychoses and are not rarely met with in normal persons.

Hallucination.—An hallucination is a false perception, entirely subjective, and not based upon any knowledge derived from without. An individual who hears voices and sees objects when none exist is the subject of hallucinations.

Imperative Conception.—This is an idea which dominates the patient's actions to a greater or less extent, although its falsity is recognized.

A morbid impulse is an irresistible desire to commit an act which the patient knows to be wrong. It is usually the result of an imperative conception. *Kleptomania* is a morbid desire to steal. *Pyromania* is a morbid desire to set fire to buildings.

Delirium.—Delirium is a mental state characterized by a rapid flight of ideas that are incoherent and often unintelligible. It may result from an infectious disease, an intoxication (alcoholism, atropin poisoning, uremia, etc.), organic cerebral disease, profound inanition, or hyperacute mania.

DISEASES OF THE BRAIN, SPINAL CORD AND NERVES

ACUTE CEREBRAL LEPTOMENINGITIS

Definition.—An acute inflammation of the pia mater and arachnoid.

Etiology.—(1) It may be a primary affection excited by the *Diplococcus intracellularis* (cerebrospinal fever) or by the pneumococcus. (2) It may be tuberculous, tubercle bacilli from a primary focus of disease elsewhere in the body reaching the meninges through the blood-vessels or lymph-channels. (3) It may follow injury, disease of the cranial bones, or otitis media (streptococcus, staphylococcus, pneumococcus). (4) It may be a complication of a specific fever—pneumonia, typhoid fever, erysipelas, influenza, etc. (the organisms exciting the primary infection or pyogenic bacteria).

Pathology.—The membranes are injected, cloudy, and more or less edematous. The subarachnoid space is distended with a seropurulent or purulent exudate. The substance of the brain may also be involved. The ventricles are often dilated and filled with turbid fluid. In some cases the process extends over the entire brain and even to the spinal cord; in others it is more or less localized to the convexity or base. The tuberculous form and that following middle-ear disease are usually basilar. In the tuberculous form, which is nearly always secondary, an infiltration of yellowish, gelatinous material is found at the base, especially about the optic chiasm. Small tubercles can usually be detected along the blood-vessels in the Sylvian fissures. The amount of fluid in the lateral ventricles is often considerably increased (acute hydrocephalus).

Symptoms.—The onset may be sudden or insidious. Headache, severe and persistent, is rarely absent. Vomiting is often a prominent symptom, especially in basilar meningitis. It frequently occurs independently of the presence of food in the stomach. The temperature is moderately high (102° – 104° F.) and very irregular. The pulse is sometimes infrequent (70 to 50 a minute). The bowels are constipated and the abdominal wall is retracted. Facial herpes is common in the meningococcic form. Symptoms of cerebral irritation soon develop. These comprise delirium, contraction of the pupils, photophobia, intolerance to sound, general hyperesthesia, Kernig's sign, muscular twitchings, and, perhaps, convulsions.

When the exudate is sufficient in amount to exert marked pressure, paralytic phenomena develop. Palsies, gross or localized, take the place of convulsions; coma follows delirium; the pupils dilate and the eyeballs roll up; photophobia is replaced by blindness, and intolerance to sound by deafness. If the finger be drawn across the body, a bright-red line develops and lingers for some minutes; this is the *tâché cérébrale* of Trousseau. The pulse now becomes rapid, weak and irregular; the respiration often acquires the Cheyne-Stokes type, and the temperature falls, often becoming subnormal.

When the process involves the base, retraction of the head with rigidity of the back of the neck, optic neuritis, and paralysis of the cranial nerves are prominent symptoms. Fluid obtained by lumbar puncture is frequently turbid and may contain the organism provocative of the disease.

Diagnosis.—In *tuberculous meningitis* the onset is usually insidious, symptoms of ill health preceding the outbreak for days or weeks; the symptoms of a basilar involvement are marked; tuberculous lesions may be detected elsewhere in the body; tubercles are occasionally seen on the choroid, and finally fluid obtained by lumbar puncture is usually clear and contains an excess of lymphocytes and tubercle bacilli.

Cerebrospinal Fever.—In this disease spinal symptoms—opisthotonos, pain in the back, and contractures—are usually

marked; there may be a purpuric rash; and the fluid obtained by lumbar puncture is turbid, is rich in polymorphonuclear leucocytes, and contains meningococci.

Typhoid Fever.—This may be recognized by the regular fever, roseolar rash, abdominal symptoms, and the Widal reaction. The severe cerebral symptoms—delirium, spasms, and retraction of the head—now and then observed in typhoid fever are usually due to cerebral congestion or to the toxemia, and only very rarely to meningitis.

Prognosis.—The outlook is grave. The tuberculous and purulent forms, and that following pneumonia are almost invariably fatal. The duration is usually from a few days to two or three weeks, but the meningococcic form occasionally becomes chronic and persists for several months. Even if recovery occurs, blindness, paralysis, or mental impairment may remain as a permanent sequel.

Treatment.—This should be conducted on the same lines as that of cerebrospinal fever. The patient should be placed in a quiet, well-ventilated room. An ice-bag should be applied to the head. In the robust, wet cups or leeches may be applied to the neck. Blisters are objectionable. The diet must be liquid. Constipation should be relieved by enemas. Restlessness, headache, and convulsions will call for bromids, chloral, phenacetin, or morphin. Warm baths and lumbar puncture are also of service. In the meningococcic form serum treatment should be instituted as soon as possible. If middle-ear disease is the exciting factor and the symptoms can be definitely localized, operation may be justifiable.

CHRONIC CEREBRAL LEPTOMENINGITIS

Definition.—A chronic inflammation of the pia mater.

Etiology.—It may result from syphilis, alcoholism, traumatism, or sunstroke. It may be secondary to acute infectious leptomeningitis. It is an associated condition in abscess and tumors of the brain.

Symptoms.—These comprise persistent, dull headache, mental deterioration, vertigo, muscular weakness, a low grade of optic neuritis, and occasionally nausea, vomiting, and tinnitus. Acute exacerbations are not infrequent, and are characterized by fever, severe headache, delirium, convulsions, and stupor.

Diagnosis.—*Cerebral Tumor.*—In tumor the symptoms are more severe and of a more focal character, and the optic neuritis is of a high grade.

Uremia may be recognized by the previous history, occurrence of chronic nephritis, albuminuric retinitis, etc.

Prognosis.—The outlook is very uncertain. A complete cure is sometimes obtained in syphilitic cases when specific treatment is instituted early.

Treatment.—In syphilitic meningitis mercury and potassium iodid should be used freely. In other instances courses of ergot and potassium bromid are occasionally useful. Applications of the thermocautery sometimes give relief. Tonics and somnifacients are frequently indicated.

CHRONIC CEREBRAL PACHYMENINGITIS

Definition.—Inflammation of the dura mater.

Etiology.—Inflammation of the external layer may result from injury, syphilis, or caries of the bone. Inflammation of the internal layer (hemorrhagic pachymeningitis) is most commonly met with in chronic insanities. Less frequently it follows trauma of the head or sunstroke, or occurs in chronic alcoholism, severe anemia, or chronic disease of the blood-vessels.

HEMORRHAGIC PACHYMENINGITIS

(Hematoma of the Dura Mater)

This condition is characterized by the formation of layers of delicate connective tissue extraordinarily rich in thin-walled blood-vessels from which the blood is prone to escape, producing hematomata of various sizes.

Symptoms.—In some cases there are no manifestations during life.

If the condition is marked, the following phenomena may be observed: Headache, failure of memory, impairment of intellect, stupor, contracted pupils, local convulsions, or palsies. The symptoms may alternately improve and grow worse over a long period. In grave cases, associated with extensive hemorrhagic effusion, the symptoms resemble those of cerebral apoplexy. The treatment is symptomatic.

CHRONIC HYDROCEPHALUS

(Congenital Internal Hydrocephalus; Water on the Brain)

Definition.—A condition in which there is an excessive accumulation of fluid in the ventricles of the brain.

Etiology.—The disease usually begins at birth or within the first few months of extra-uterine life. Occasionally, it is acquired after the first two years. The immediate cause of the excess of fluid is either an obstruction to the outflow from the ventricles, usually at the aqueduct of sylvius (*obstructive hydrocephalus*) or, much less frequently, excessive secretion of fluid or defective absorption as a result of adhesions in the spinal subarachnoid space (*communicating hydrocephalus*).

Pathology.—The head is large and round; the bones are thin and translucent; the sutures and fontanelles are enlarged, and, if life has been prolonged, are filled with numerous Wormian bones. The convolutions of the brain are flattened and the sulci more or less obliterated. The ventricles are greatly distended with a watery fluid of low specific gravity, containing a trace of albumin. The ependyma is often thickened and roughened. Malformations are frequently observed, and probably result from the causes which induced the effusion.

Symptoms.—Sometimes the disease develops before birth, and the large head interferes with the delivery of the child. In other cases nothing peculiar is observed until the child is several months old, when the swelling of the head attracts the attention of the parents. The head assumes a globular shape; the fontanelles and sutures remain open; the face becomes relatively small; the eyes protrude and are directed downward

from the pressure of the fluid on the supra-orbital plates; the scalp is thin and stretched; the superficial veins are distended; and the hair is often scanty. In some cases the head is so heavy that the thin neck can no longer support it, and it falls forward on the breast.

As a rule, the intelligence is considerably impaired, but exceptional cases are marked by precociousness. Motor phenomena are frequently present; the reflexes are exaggerated; one or more of the members may be the seat of a spastic paralysis; and convulsions develop in many cases.

The duration varies in different cases. The large majority soon die of inanition, convulsions, or some intercurrent disease to which their reduced vitality makes them an easy prey; but in a few cases life is prolonged for many years.

Diagnosis.—Hydrocephalus must not be mistaken for *rachitic enlargement of the head*. In the latter the head is square instead of globular; the intelligence is good; there are no motor phenomena; and enlargements are usually detected at the ends of the long bones and at the junction of the cartilages with the ribs.

Prognosis.—The outlook is unfavorable. Occasionally arrest of the disease occurs spontaneously or results from aspiration of the fluid.

Treatment.—The treatment is unsatisfactory.

Spinal puncture is sometimes of benefit when there is free communication between the ventricles and the subarachnoid space, and, rarely, puncture of the ventricles or corpus callosum and withdrawal of fluid or a decompressive operation affords some relief in the obstructive form.

PARETIC DEMENTIA

(General Paralysis of the Insane; General Paresis; Chronic Meningo-encephalitis)

Definition.—Chronic meningo-encephalitis of syphilitic origin, characterized clinically by progressive mental deterioration and general paresis.

Etiology.—The disease occurs most frequently between the ages of 35 and 50 years. It is much more common in men than in women. Syphilis is the essential etiologic factor. Mental strain, alcoholism and cranial injury may serve as auxiliary causes.

Pathology.—The membranes are opaque, thickened, and at places adherent to the brain substance. The cortex, especially of the frontal lobes, is more or less atrophied and increased in firmness. Microscopic examination reveals a marked increase in the neuroglia, with numerous spider cells, thickening of the vessel-walls, distention of the perivascular spaces with lymphocytes, and degeneration of the ganglion-cells and atrophy of the nerve-fibers.

In some cases degenerative changes are also observed in the spinal cord, especially in the dorsal columns (*tabo-paralysis*).

Symptoms.—The disease usually begins insidiously with a change in disposition: the industrious become slothful; the ambitious, apathetic; the chaste, dissolute; the liberal, parsimonious; the complaisant, churlish; and the truthful, false. The energy relaxes, the judgment weakens, and the memory fails. As the faculties become impaired, a peculiar egotism and mental exaltation develop; the patient becomes boastful, loquacious, and easily provoked to furious outbreaks. The failure of memory is early noted in writing by the use of wrong letters and the suppression of syllables. At this time motor phenomena may be observed; the tongue trembles when it is protruded; the speech is slow, hesitating, and indistinct; the pupils are often unequal; and the gait is somewhat shuffling.

The most characteristic psychic symptom of fully developed parietic dementia is the delusion of grandeur, which is shown in the patient's exaggerated estimate of his social or political status, wealth, strength, or intellectual power. The mind is usually serene and cheerful, but periods of profound depression are not infrequent, and occasionally, there is throughout emotional depression with delusions of persecution or of physical illness. The sensibilities are blunted and the animal

nature is emphasized. The mind becomes more and more involved; there is extreme indifference to all that transpires; the appetite is voracious, and in eating the patient bolts his food and soils his clothes. The tremor of the tongue increases, and spreads to the lips and other parts of the face; the speech is indistinct and "scanning;" the pupils fail to respond to light, but still accommodate for distance (Argyll-Robertson pupil); and the reflexes are usually increased, though they may be lost. Seizures of an epileptiform or apoplectiform character are not uncommon. The cerebrospinal fluid contains a considerable amount of albumin (globulin) and an excess of lymphocytes, and yields a positive Wassermann reaction and positive colloidal gold reaction.

In the final stage mental power is almost obliterated; the health fails; the bladder and rectum become unretentive; the unsteadiness of gait increases; and at last the patient is unable to leave his bed. Death usually results from exhaustion or intercurrent disease.

Diagnosis.—The insidious change in disposition, failure of memory, indistinct speech, tremors, Argyll-Robertson pupil, delusions of grandeur, positive Wassermann reaction, and spinal lymphocytosis are the diagnostic features.

Interstitial Cerebral Syphilis.—Severe headache, worse at night, somnolence, persistent localized paralyses, especially of the cranial nerves below the third, early emaciation, and absence of the Argyll-Robertson pupil, of generalized tremors, and of delusions of grandeur are in favor of cerebral syphilis.

Prognosis.—The outlook is virtually hopeless. However, remissions or lucid intervals lasting several months or even years are not infrequent. The average duration is about three years.

Treatment.—This includes the avoidance of all mental and physical excitement, and the employment of the general measures that are of service in improving nutrition. Vigorous antisypilitic treatment is sometimes followed by clinical improvement, especially in early cases. For obvious reasons

the treatment is best carried out in a well-ordered sanatorium or asylum.

CEREBRAL PARALYSIS IN CHILDREN

(Spastic Paralysis of Infants)

Definition.—Hemiplegia, diplegia, or paraplegia appearing at birth or in the first few years of life, and dependent upon defective development (agenesis) or acquired disorder of the upper motor neurons.

Pathology.—After death one of the following conditions is found: Porencephalus (cystic condition of the brain), atrophy and sclerosis of the convolutions, meningo-encephalitis, or meningeal hemorrhage. The primary lesion in *prenatal* cases is not definitely known, but it is probably a circulatory one; in *congenital* cases it is usually meningeal hemorrhage induced by difficult labor; and in *infantile* cases it may be acute encephalitis, hemorrhage, thrombosis, or embolism, the result of an antecedent infection.

Symptoms.—In the *hemiplegic* variety the onset is sudden, and is frequently attended with fever, convulsions, or coma. After a few hours or days these severe symptoms subside, and the child is left paralyzed on one side of the body. In rare instances the paralysis ultimately disappears and the child is restored to health, but in the large majority of cases it persists and is followed by secondary rigidity. Imbecility, epilepsy, and choreiform or athetoid movements in the affected members are very common sequelæ.

The *diplegic* or *paraplegic* form usually dates from birth, and is characterized by rigidity and loss of power in the arms and legs, or in the legs alone. Children thus affected are generally idiots or imbeciles. Choreiform or athetoid movements are frequently present. Epilepsy is also common.

The term *Little's disease* is applied to a pure form of spastic paralysis, especially of the legs, occurring at birth or in infancy, and resulting from defective development of the

pyramidal tracts. It differs from the ordinary form of cerebral diplegia due to circulatory lesions or encephalitis in the absence of mental defects, epilepsy and athetoid movements.

Treatment.—During the convulsive stage an ice-bag should be applied to the head and chloral or bromid administered by the mouth or rectum. In congenital cases, when there are bulging of the fontanel, asphyxia, and convulsions, operation should be considered. The paralysis usually resists treatment; but subsequent rigidity may be lessened by massage and passive movements, and the deformity by tenotomies or by mechanical appliances. Division of the posterior nerve-roots has also been practiced with some success in overcoming spasticity in the legs.

CEREBRAL HEMORRHAGE

(Cerebral Apoplexy)

Etiology.—The affection is most commonly met with in persons between 45 and 60 in whom the arteries are degenerated, but it may occur at any period, even in childhood. All causes that lead to arterial disease, such as gout, syphilis, alcoholism, and chronic nephritis, predispose to it. Chronic nephritis owing to the occurrence of arteriosclerosis, high vascular tension, and cardiac hypertrophy is especially dangerous. Heredity predisposes in as much as members of certain families are more prone than others to sclerosis of the vessels.

The attack may be precipitated by mental or physical excitement, alcoholic excess, or some reflex disturbance, as gastric irritation. In children it may be excited by a paroxysm of whooping-cough or by a convulsion. Occasionally, it occurs after diphtheria or scarlet fever, the toxins of these diseases producing degenerative changes in the arterial walls.

Pathology.—In children the hemorrhage is most commonly cortical; in adults it is usually within the brain-mass. The blood-vessels are usually atheromatous, and are sometimes the seat of miliary aneurysms. The hemorrhage varies

greatly in quantity; sometimes it is small—merely a capillary oozing; at other times it may flood an entire hemisphere. Its most common seat is the internal capsule—the motor highway between the optic thalamus and the corpus striatum. In recent hemorrhages the clot is dark and soft, and the surrounding tissue stained and more or less lacerated. If the hemorrhage has not been very copious, the clot loses its color, shrinks, and is finally absorbed, and the damaged cerebral fibers are replaced by proliferated connective tissue, which contracts and forms a scar more or less pigmented with hematoidin. In other cases, instead of a scar, a cyst is formed enclosing a clear, straw-colored fluid.

Large effusions are followed by secondary changes, which are systemic and extend in the direction in which the affected nerve-fibers transmit impulses; that is, toward the periphery, if the fibers are motor, and toward the nerve-centers if they are sensory. After an extensive lesion of the internal capsule secondary degeneration of the motor tracts soon begins and may be traced downward into the spinal cord.

Symptoms.—*Prodromal symptoms* indicating cerebral congestion sometimes precede the attack; these are headache, vertigo, disturbed sleep, tinnitus aurium, and, perhaps, a sense of numbness or weakness on the side that is to be affected. An attack of vomiting occasionally precedes the hemorrhage.

The Attack.—In many cases the patient falls suddenly unconscious without previous warning. The face is flushed; the eyes are injected; the lips are blue; the breathing is stertorous; the pulse is full and slow; the temperature is at first subnormal from shock, but later it is elevated from irritation; and the urine and feces may be passed involuntarily. Convulsive seizures are not infrequent; they result from irritation transmitted to the undamaged motor regions. Even while the patient is comatose, the paralysis may be detected. The head and eyes are sometimes strongly rotated toward the side of the hemorrhage (conjugate deviation); one cheek often flaps more than the other; the pupils may be unequal; any

movements that the patient may make are restricted to the sound side; when the affected arm is raised and let fall, it drops lifeless; and occasionally the temperature is higher in the axilla of the paralyzed side. In grave cases the patient does not awake from the coma; the pulse grows feeble; the respirations acquire the Cheyne-Stokes type; the reflexes are abolished; mucus collects in the throat and produces a rattling sound; the temperature rises to 102° or 103° ; and death results after the lapse of a few hours or one or two days.

In certain cases the paralysis rapidly sets in, but unconsciousness develops gradually and does not become complete for twenty-four hours (ingravescent apoplexy). In other cases of cerebral hemorrhage loss of consciousness is transient or wanting.

Subsequent Symptoms.—If the attack does not prove fatal, consciousness is usually restored in from twelve to forty-eight hours, and if the hemorrhage is in its usual location, there remains a hemiplegia on the opposite side. The muscles of the upper part of the face and thorax, however, usually escape, because they are accustomed to act in unison with their fellows on the opposite side, and such muscles appear to be innervated from both sides of the brain. When the tongue is protruded, it deviates toward the paralyzed side. The deep reflexes are exaggerated on the affected side and irritating the sole of the foot causes extension of the great toe (Babinski's sign). Aphasia is common with right hemiplegia. There is no tendency to rapid wasting of the affected muscles. Sensation is unimpaired unless the entire posterior limb of the internal capsule is involved, when there is hemianesthesia with the hemiplegia. The gait is peculiar; in walking the patient supports the paralyzed arm and swings the leg forward by a rotary movement imparted to it by the trunk. If the clot has been small, the paralysis may completely disappear. More frequently, recovery is only partial. The power of the facial muscles is, as a rule, restored entirely, and the leg usually improves more than the arm.

In unfavorable cases the muscles become rigid from a degenerative process traveling down the direct and crossed pyramidal tracts of the spinal cord; this condition is indicative of permanent disability.

Mental symptoms, especially impairment of memory and loss of emotional control, frequently follow the attack.

Diagnosis.—The coma of apoplexy must be distinguished from that of *uremia*, *opium-poisoning*, *alcoholism*, *diabetic acidosis*, *sunstroke*, and *fracture of the skull*. The absence of the characteristic features of these conditions together with the age of the patient, the state of his arteries, and the signs of hemiplegia will usually prevent an error in diagnosis.

Embolism.—This usually occurs in earlier life; it is commonly associated with valvular disease, especially mitral lesions; premonitory symptoms are rarely present; and disturbances of the pulse, temperature, and respiration are usually slight or wanting.

Thrombosis.—This also produces hemiplegia, but its development is more often gradual, premonitory symptoms, especially transient aphasia or weakness in the members afterwards paralyzed, is much more common than with cerebral hemorrhage, coma is often absent, and changes in the pulse, respiration and temperature are relatively infrequent.

Fracture of the Skull.—In this condition the diagnostic points are the history or the evidences of local injury, bleeding from the nose or ears, and the occurrence of ecchymosis about the eyes or mastoid processes.

Opium Poisoning.—The extreme contraction of the pupils, the very infrequent respiration, and the absence of paralysis are the suggestive features.

Alcoholism.—In this condition the coma is rarely complete, signs of hemiplegia are absent, and the odor of alcohol is usually present on the breath.

Uremia.—Signs of nephritis are present, convulsions are much more common and paralysis is much less common than in cerebral hemorrhage, albuminuric retinitis may be found,

and the blood usually shows an excess of non-protein nitrogen.

Diabetic coma may be recognized by the fruity odor of the breath, the presence of sugar and acetone bodies in the urine and the absence of hemiplegia.

Prognosis.—This is always uncertain. Persistent unconsciousness, high temperature, loss of reflexes, and embarrassed respiration are unfavorable phenomena. If the attack does not prove fatal, there is always danger of recurrence, since the etiologic conditions still remain.

Treatment.—*Prophylaxis.*—Patients predisposed to apoplexy should lead a quiet life, free from mental and physical excitement. The diet should be nutritious, but easily digestible. Constipation should be relieved by the occasional use of a saline laxative. To secure a free return of the blood from the brain the clothes at the neck should be loose.

The Attack.—The head and shoulders should be slightly elevated, and an ice-bag applied to the head. Croton oil (1 to 3 drops) in a little glycerin or olive oil may be placed on the back of the tongue to secure prompt catharsis. If the pulse is strong, venesection is indicated and should be continued until the pulse softens. Bleeding cannot undo the damage already done, but by relieving cerebral congestion, it may serve to arrest bleeding that is still in progress or to prevent an early recurrence. On the other hand, when the face is pale and the pulse feeble, diffusible stimulants such as camphor and ether should be given very cautiously. If collections of mucus interfere with breathing, the patient should be gently turned on his side and the mucus removed.

To prevent the formation of bed-sores the position should be frequently changed and the parts subjected to pressure thoroughly cleansed.

Subsequent Treatment.—Even in the mildest cases the patient should not be allowed to leave his bed for two or three weeks, and during this time the diet should be light and unstimulating. After the acute symptoms have entirely disappeared, which will rarely be earlier than ten days or

two weeks after the attack, massage should be systematically practiced. It aids in the restoration of power and in the prevention of contractures. After the lapse of three or four weeks triweekly applications of the faradic current may be of service. Strychnin is often given at this time, but it probably exerts no other influence than that of a general tonic. In some cases warm saline baths (90°-95° F.) combined with passive movements prove useful adjuvants.

OBSTRUCTION OF THE CEREBRAL ARTERIES

(Embolism; Thrombosis)

Etiology.—*Cerebral emboli* may be derived from the valves of the heart in endocarditis; from an atheromatous plate in the aorta; or from a thrombus in the heart or in the sac of an aneurysm. Obstruction from embolism may occur at any age, but it is much more frequently observed in young adults than at the extremes of life.

Cerebral thrombi are usually caused by arteriosclerosis or syphilitic endarteritis. They are most frequently observed in old persons, but those dependent upon syphilis often occur in early adult or middle life.

Pathology.—*Emboli* are most frequently found in a branch of the left middle cerebral artery or of the sylvian artery. If the artery obstructed is a large one, infarction of the brain with softening ensues. If the area affected is small, absorption of the dead tissue usually follows, with the formation of a cicatrix. Infective emboli give rise to abscesses.

Thrombi are usually found in the middle cerebral artery, basilar or vertebral arteries and produce the same lesions as emboli.

Symptoms.—*An embolus* lodging in the middle cerebral artery usually causes abrupt hemiplegia and, if on the left side of the brain, motor aphasia. There are usually no cerebral prodromes, consciousness is not often lost, and marked disturbances of the temperature, respiration and pulse are uncommon.

If the basilar artery is obstructed, there may be extensive paralysis on both sides of the body, and later symptoms of bulbar disease—namely, paralysis of the lips, pharynx, and esophagus, disturbance of the heart, and Cheyne-Stokes breathing.

In *thrombosis* the symptoms are similar to embolism, but they develop more slowly, and are frequently preceded by prodromes indicating disturbed cerebral circulation, such as headache, vertigo, and numbness or transient weakness in the members that are subsequently paralyzed. There is a very marked tendency to recurrence of attacks.

Subsequent Symptoms.—In both embolism and thrombosis, if the artery obstructed has been large, the paralysis is likely to persist and to be followed by symptoms of cerebral softening—namely, failure of memory, vertigo, headache, disturbed sleep, irritability, and finally dementia.

Diagnosis.—The differential diagnosis between cerebral embolism or thrombosis and *hemorrhage* has already been considered (see p. 470).

Prognosis.—The outlook is always serious, although recovery from the attack itself often occurs both in embolism and thrombosis.

Treatment.—After obstruction from embolism the patient should be kept at absolute rest for a week or two, and subsequently the paralysis treated as after apoplexy. In thrombosis treatment is rarely of avail; in syphilitic cases, however, active antiluetic treatment should be instituted.

TUMORS, INFECTIOUS GROWTHS AND CYSTS OF THE BRAIN

Etiology.—The etiology of brain tumors is obscure. Males are more frequently affected than females. No age is exempt, but the majority of brain tumors occur between the ages of 15 and 40 years. Occasionally the history of some remote injury is obtainable. Heredity has little or no influence.

Varieties.—Glioma, endothelioma, sarcoma, carcinoma, tuberculoma, and gumma are the most common varieties. Occasionally, cholesteatoma, fibroma, angioma and parasitic cysts are observed.

Glioma.—This tumor is found almost exclusively in the brain. It arises from the neuroglia and may be soft like brain-substance or firm like fibrous tissue. It is observed chiefly in young persons. It is most commonly found in the cerebral hemispheres and it grows slowly.

Endothelioma.—This is a benign, slowly growing tumor, arising from the meninges, blood-vessels or lymphatics. It rarely infiltrates the brain and usually shells out easily.

Sarcoma as a primary tumor arises from the membranes and is usually circumscribed. Secondary sarcomata are usually found within the brain and are, as a rule, multiple.

Carcinoma is almost invariably secondary and is often multiple.

Tuberculoma is observed most frequently in childhood. It may develop on the meninges or in the substance of the brain.

Gumma usually occurs as a more or less circumscribed caseous meningitis at the base of the brain or the convexity of a hemisphere. Solitary gummata within the brain are comparatively rare.

Symptoms.—GENERAL SYMPTOMS.—(1) Headache is rarely absent; in superficial growths it is sometimes localized and associated with tenderness. (2) Vomiting is a common symptom, especially in tumors at the base of the brain; it is often unassociated with nausea, and does not relieve the attending headache. (3) Choked disc is present in about 80 per cent. of the cases. (4) Vertigo is often marked, especially in tumors of the basal ganglia and cerebellum. (5) Convulsions, local (Jacksonian epilepsy) or general, occur in about 50 per cent. of all cases. (6) Psychic phenomena—failure of memory, depression of spirits, varying degrees of dementia, irritability of temper, and emotional states—are not infrequently present.

Insomnia, changes in the rate and rhythm of the pulse, polyuria, and glycosuria are occasional symptoms.

FOCAL SYMPTOMS.—These depend entirely upon the location of the tumor. The following are the chief localizing symptoms:

Prefrontal Region.—Mental torpor, irritability, loss of memory, and change in disposition frequently occur. Pressure on the precentral convolutions may cause motor aphasia, on the optic nerves, visual disturbance, and on the olfactory nerves, loss of smell.

Precentral Convolutions.—If the tumor irritates the centers, local spasms (Jacksonian epilepsy) develop; if it exerts enough pressure to destroy function, paralysis results. Motor aphasia may also occur.

Parietal Lobes.—Tumors in this region frequently cause disturbances of cutaneous and deep sensibility, especially a loss of power to recognize objects by handling them (astereognosis).

Occipital Lobes.—Hemianopsia is common, and there may be psychic blindness if the growth is on the left side. Word-blindness may also result from pressure on the angular gyrus.

Temporal Lobes.—Only in tumor of the left side are there likely to be definite symptoms—word-deafness and uncinat fits (“dreamy state” with involuntary movements of the lips and tongue and subjective disturbances of taste and smell).

Corpus Callosum.—Tumors in this region may result in mental dulness, slowly developing diplegia, and an inability to execute simple movements when the patient wishes to do so, while the power to make them on occasion still remains (motor apraxia).

Corpus Striatum.—Tumor of this body usually produces symptoms referable to involvement of adjacent structures, especially hemiplegia, and if on the left side, motor aphasia.

Optic Thalamus.—Tumor of the thalamus is likely to cause spontaneous pains, flaccid hemiparesis (pressure on internal capsule), hemiataxia, hemichorea or hemiathetosis—all on the

opposite side. Hemianopsia and partial deafness may also occur.

Cerebral Peduncle.—Tumor in a peduncle may produce "alternate hemiplegia," that is, hemiplegia of the opposite side and palsy of the third nerve on the side of the lesion.

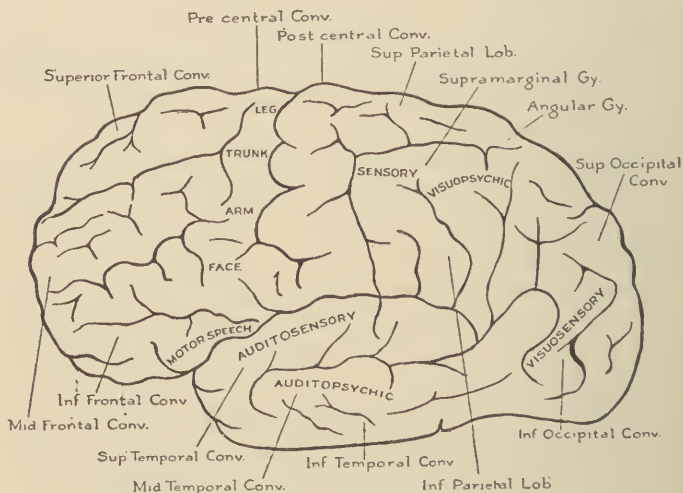


FIG. 16.—Functional areas of cerebral cortex of the left side (after Campbell).

Corpora Quadrigemina.—Tumor of these bodies cause oculo-motor palsies, nystagmus, choked disc, and disturbance of equilibrium (involvement of cerebellar peduncle).

Pons.—Pontile tumors cause paralysis of certain cranial nerves, especially the facial, trigeminus or abducens, on one side and hemiplegia on the other. Paralysis of the lateral associated movements of the eyes is also common (Spiller).

Pituitary Body.—Tumor in this structure may cause acromegaly or gigantism, or the Fröhlich syndrome—adiposity, somnolence, increased sugar tolerance, and changes in the secondary sexual characters. Bi-temporal hemianopsia is also common.

Cerebellum.—The most common indications of cerebellar tumors are occipital headache, choked disc, vomiting, vertigo, hypotonia, and asynergy, which is shown by a reeling, staggering gait, inability to repeat rapidly pronation and supination of the hand or similar movements (adiadokokinesis), and mis-measurement of movements (dysmetry). Nystagmus is also common.

Diagnosis.—*The Character of the Growth*.—This cannot always be determined. The early age, the rapid progress, and the antecedent history may suggest *tubercle*. The early age, slow progress, and mild pressure-symptoms may suggest *glioma*. The history, age, and concomitant symptoms will often indicate *syphilis*. The presence of a primary growth will lead to the diagnosis of *malignant disease*.

Abscess.—Cerebral tumor must be distinguished from abscess. The latter usually results from traumatism or is secondary to a focus of suppuration in some other part of the body; its progress is usually more rapid; optic neuritis is less common; and there is often febrile disturbance, with leukocytosis.

Chronic Meningitis.—In this affection the symptoms indicate a diffuse lesion; disturbances of temper, memory, and sleep are more marked; optic neuritis is not frequent.

Prognosis.—The outlook is always grave. If the tumor is not gummatous and is not suitable for operative intervention, the prognosis is absolutely unfavorable. The duration is from a few months to several years.

Treatment.—Antiluetic treatment should be tried in every case the nature of which is uncertain. Operative intervention is indicated when the tumor is in an accessible region (surface of cerebrum, lateral lobe of cerebellum, side of cerebellum and pons, hypophysis) and is clearly not metastatic. In skillful hands the mortality is less than 10 per cent. Even if removal is out of the question "decompression" (removal of a piece of bone with opening of the dura) may relieve the pressure symptoms, especially severe headache and choked disc. Radium

or x-ray therapy occasionally gives good results after craniectomy. Except in syphilitic growths, medical treatment is only palliative. Cold applications to the head, dry or wet cupping, and the administration of bromids, acetphenetidin, and morphin are often required to relieve pain.

ABSCESS OF THE BRAIN

(Suppurative Encephalitis)

Etiology.—(1) Cerebral abscess may be traumatic. (2) It may be secondary to suppurative inflammation of adjacent parts, as otitis media or frontal sinusitis. (3) It may be secondary to some distant focus of suppuration, as pulmonary abscess, bronchiectasis, hepatic abscess, ulcerative endocarditis. (4) It may follow one of the infectious fevers.

Pathology.—The abscess varies in size from a pea to one large enough to fill an entire hemisphere. The surrounding tissues are hyperemic, edematous, and more or less infiltrated. In the acute form the abscess is diffuse, but in long-standing cases the pus is encapsulated by a thick fibrous sac. The temporosphenoidal lobe and the cerebellum are the most frequent sites. Abscesses secondary to distant foci of suppuration are commonly multiple.

Symptoms.—Abscesses following injury frequently run an acute course, and are characterized by high fever, rigors, headache, delirium, convulsions, vomiting, and coma.

In chronic cases the *general symptoms* are headache, tenderness of the head to percussion, irritability, mental impairment, vertigo, vomiting, stupor, pallor, and loss of flesh and strength. The temperature is variable; it may be elevated, but not rarely it is normal or subnormal. The *focal phenomena* vary with the location of the abscess and are much the same as those of tumor in corresponding situations.

Prognosis.—The outlook is always grave. If the focal symptoms indicate involvement of an accessible region, such as the motor area, temporosphenoidal lobe, or cerebellum, operative intervention offers considerable hope of success.

Treatment.—If the abscess is located in one of the regions specified, the skull should be trephined and the pus evacuated. In other cases the application of wet cups to the neck, of ice-bags to the head, and the internal use of morphin, bromid of potassium, or acetphenetidin may temporarily relieve the distress.

APHASIA

Definition.—An inability to express ideas in speech or equivalents of speech or to comprehend speech or equivalents of speech.

Varieties.—Aphasia is divided into *motor* and *sensory*. Each of these is subdivided into *cortical* and *subcortical*, according as the lesion is the center itself or in the tracts communicating with the center. Sensory aphasia is further classified as *visual* and *auditory* aphasia.

Motor Aphasia.—This is an inability to express thought in words. When the lesion is in the third frontal convolution (*cortical motor aphasia*) the power of silent talking and reading are lost as well as that of articulate speech. When the lesion is in the adjacent tracts which transmit speech impulses to the articulatory muscles (*subcortical motor aphasia*), the power of articulation alone is lost. This is the most common form of aphasia.

Sensory Aphasia.—This is an inability to understand printed or written words (*visual aphasia* or *word-blindness*), or to understand spoken words (*auditory aphasia* or *word-deafness*). The lesion is in the angular gyrus, where visual word memories are stored, or in the first temporal convolution, where auditory word memories are stored, or in one of the incoming (*subcortical*) tracts of special sense.

In *cortical visual aphasia* the patient cannot read aloud or to himself, nor can he write (*agraphia*) spontaneously or from dictation. In *subcortical visual aphasia* the patient can write spontaneously and from dictation, but he cannot read what is written by himself or others.

In *cortical auditory deafness* the patient cannot understand spoken words or write from dictation. Not being able to comprehend his own speech, he misplaces words (*paraphasia*) or talks jargon. In *subcortical auditory aphasia* the patient, though word-deaf, can speak spontaneously, read aloud, and write.

Pathology.—The lesions that produce aphasia are manifold; the most important are: Tumor, gumma, abscess, depressed fracture, embolism, thrombus, or softening in the localities that correspond to the various forms of aphasia. In right-handed subjects the lesion is on the left side of the brain; in the left-handed it may, however, be on the right side. Aphasia is not always due to organic disease; it may occur as a transient condition in congestion of the brain, in sudden fright, in the convalescence of fevers, in migraine, after epileptic seizures, and in hysteria.

Diagnosis.—Aphasia must be distinguished from *aphonia*. The latter condition is an inability to utter sounds, a power not lost in aphasia; moreover, aphonia is generally dependent upon some abnormality of the larynx or of the nerves leading thereto.

Prognosis.—This depends entirely on the cause. After apoplexy the prognosis should be guarded. In cerebral softening it is absolutely unfavorable. When aphasia develops in the young, the outlook is much more hopeful.

Treatment.—The causal condition will require attention. The patient may be instructed to speak and to interpret after the manner employed in teaching the young.

SPINAL LEPTOMENINGITIS

Acute inflammation of the spinal pia mater usually occurs as a part of cerebrospinal meningitis. As an independent condition it is very rare. Occasionally, it follows traumatism or one of the specific infections. The chief *symptoms* are pain in the back radiating along the course of the spinal nerves, moderate fever, spasm of the spinal muscles, sometimes so intense as to

cause orthotonos or opisthotonos, exaggeration of the deep reflexes, and a positive Kernig's sign. Later, from pressure of exudate, there may be hypesthesia and paresis of the limbs.

Chronic inflammation of the spinal pia mater is also rare. It may follow acute meningitis from any cause or result from trauma or syphilis. The *symptoms* are similar to those of acute spinal leptomeningitis but develop gradually and continue over a long period.

MYELITIS

Definition.—The term myelitis is applied to both inflammation and ischemic softening of the spinal cord, the two conditions presenting almost the same clinical picture.

Varieties.—The process is known as *transverse myelitis* if it involves only a relatively small vertical section of the cord; as *diffuse myelitis* if it involves a large vertical extent of the cord; as *disseminated myelitis* if there are a number of discrete foci of disease, and as *poliomyelitis* (see p. 400) if the lesions are confined to the gray matter.

Etiology.—The disease may be a sequel of one of the acute infections; it may occur after trauma; it occasionally develops during the puerperium; it is sometimes secondary to disease or injury of the spine (*compression myelitis*); very frequently, however, it is the result of syphilis.

Pathology.—In early cases the affected area is soft and hyperemic; in late cases it is firm and pale (sclerotic). In the inflammatory form microscopic examination reveals degeneration or actual disintegration of the nervous elements, dilatation of the blood-vessels, and perivascular round-cell infiltration; or if the disease has been of long duration, an overgrowth of neuroglia tissue in place of the nervous elements which have been destroyed (sclerosis). In the syphilitic form the first change is a gummatous infiltration of the cord; or, more commonly, an area of ischemic necrosis (softening), the result of chronic endarteritis, and, perhaps, obstructive thrombosis. Ultimately, as in other forms of myelitis, pro-

liferated neuroglia tissue is seen in place of cells and fibers that have undergone liquefaction.

Symptoms.—*Acute Transverse Myelitis.*—The onset is marked by numbness in the limbs, and sometimes by pain in the back and a girdle sensation. Moderate fever is sometimes present. Paralysis and anesthesia for all forms of sensation quickly develop in the parts below the level of the lesion. The paralysis is flaccid and atrophic in the muscles supplied by the diseased segments, and flaccid or spastic in the muscles innervated by segments below the level of the disease, according as the latter completely or partially interrupts the conductivity of the cord. In the lower limbs, even if the paralysis is flaccid at first, it usually becomes spastic in the course of a week or two if the patient survives. The sphincters of the bladder and rectum are usually affected, sexual power is often abolished, and bed-sores often develop with great rapidity over the buttocks and heels. Atrophy and reactions of degeneration occur in the muscles supplied by nerves arising from the diseased segment.

The outlook is always serious. More or less disability usually remains after the subsidence of the acute symptoms. Complete recovery may occur, however, in mild cases. Not rarely death ensues from cystitis and pyelonephritis, bed-sores, or paralysis of the respiratory musculature.

Acute ascending myelitis is characterized by progressive loss of motion and sensation beginning in the legs and rapidly ascending until the respiratory musculature is affected. Death frequently occurs at the end of a few days from asphyxia, or at a later period from cystitis, bed-sores, or hypostatic pneumonia.

Disseminated myelitis is rare. It produces palsy of isolated groups of muscles and irregularly distributed areas of anesthesia. In the later stages of the disease the clinical picture may be that of multiple sclerosis.

Compression myelitis is characterized by localized and deep-seated pain in the back, rigidity of the spinal column,

angular deformity, stabbing pains radiating to the limbs or around the trunk (girdle pains), spastic paralysis below the level of the lesion, impairment of sensation, and disturbance of the sphincters. X-ray studies are often a valuable aid to diagnosis.

Chronic Myelitis.—This term is often applied to the chronic stage of acute myelitis and to compression paraplegia, the result of vertebral disease or spinal tumor. As a slowly progressive condition it is occasionally met with in syphilis. The chief feature is slowly developing spastic paraplegia with exaggeration of the reflexes, more or less sensory disturbance, usually paresthesia or hypesthesia, and sphincter disorders. The last not rarely precede the other symptoms by several months.

Diagnosis.—*Acute Poliomyelitis.*—In this disease the bladder and rectum are not affected and there is no anesthesia.

Landry's Disease or Acute Ascending Paralysis.—In this affection there is an absence of sphincteric disturbance and of muscular wasting, and if sensory disorders are present, they are slight.

Multiple Neuritis.—The sphincters are not affected; bed-sores are rare; the pains are referred to peripheral parts of the limbs, and the nerve-trunks are tender on pressure.

Meningitis.—In this disease hyperesthesia, pain, rigidity of the back, tonic muscular spasms, and Kernig's sign are prominent in the clinical picture, while paralysis is less pronounced, the sphincters escape, and bed-sores are usually absent. Moreover, in meningitis characteristic changes usually occur in the spinal fluid.

Hemorrhage into the Cord.—In this condition the paralysis develops abruptly.

Treatment.—If possible, the patient should be placed on a water-bed or air-bed. Counterirritation should be avoided on account of the danger of bed-sores. Cold, however, in the form of Chapman's ice-bags may be applied to the spine. Daily warm baths (90° F.) lasting about ten

minutes are useful. When there is suspicion of syphilis, mercury or iodids should be given a thorough trial.

Every precaution should be taken against the development of bed-sores. Frequent change of the patient's position, absolute cleanliness of the parts subjected to pressure, and bathing with alcohol and water will do much toward obviating this complication. Retention of urine must be met by systematic catheterization under the most strict aseptic precautions. When there is constant incontinence, a carefully adjusted urinal should be employed.

Any tendency to cystitis will call for daily irrigation of the bladder with a solution of boric acid or other mild antiseptic solution. After the acute symptoms have entirely subsided, passive movements, massage, and electricity should be employed to maintain the nutrition of the affected muscles and to prevent fixation of the joints.

CHRONIC ANTERIOR POLIOMYELITIS—PROGRESSIVE SPINAL MUSCULAR ATROPHY

Definition.—A chronic disease characterized anatomically by atrophy of the nerve cells in the anterior gray horns of the spinal cord, and manifested clinically by a progressive wasting of the muscles and a corresponding loss of power.

Etiology.—The disease is more common in males than in females. It occurs most frequently in adults between the ages of twenty and fifty. However, a rare hereditary and familial form occurs in early childhood. The etiology is obscure. Syphilis is sometimes a factor.

Pathology.—Microscopic examination of the cord reveals atrophy or complete disintegration of the ganglion cells in the anterior cornua and an overgrowth of the neuroglia. The anterior nerve-roots, peripheral motor nerve-fibers, and affected muscles also show degenerative atrophy. In addition to these lesions, there is often sclerosis of the anterolateral white tracts (amyotrophic lateral sclerosis).

Symptoms.—*Duchenne-Aran Type.*—The onset is insidious. The muscles of the hand usually suffer first. The thenar and the hypothenar eminences and the interosseous muscles become more flaccid than normal and gradually waste. Accompanying the atrophy there is a corresponding loss of muscular power. When the interossei no longer afford opposition to the long flexor and extensor muscles, the hand assumes a claw-like position (*main en griffe*), which is quite characteristic. Fine fibrillary tremors or twitchings are almost invariably present in the affected muscles. After the lapse of months, perhaps years, the wasting and paresis spread to the muscles of the shoulder and arm, and then to the neck and trunk. The legs are usually not involved until late, and often escape entirely. Occasionally, however, the disease begins in the lower extremities or back, but this is rare. In the late stages the patient may be reduced to a mere skeleton. In many cases the process invades the medulla, in which case the symptoms of *bulbar palsy* are superadded.

There may be some complaint of coldness or of dull pain, but sensation is not impaired. The deep reflexes are lost in the affected limbs and the paralyzed muscles remain flaccid. The reactions of degeneration are sometimes present, but more often there is simply diminished response first to the faradic and then to the galvanic current. The sphincters are not involved.

Werdnig-Hoffman Type.—In this rare form, which is familial and hereditary and develops in early childhood, the atrophy begins in the muscles of the thigh and pelvic girdle, and then spreads to those of the arms and legs.

Diagnosis.—Chronic poliomyelitis must be distinguished from other conditions causing slowly progressing atrophy and weakness, such as amyotrophic lateral sclerosis, muscular dystrophy, multiple neuritis, and syringomyelia.

Amyotrophic Lateral Sclerosis.—In this disease the atrophy is associated with spastic symptoms and the tendon-reflexes are exaggerated.

Muscular Dystrophy.—This is commonly an hereditary or a family affection. It occurs in childhood and attacks primarily large muscles (calf, shoulder girth, or face). There is no fibrillary twitching and the reactions of degeneration are never present.

Multiple Neuritis.—In this disease the paralysis precedes the atrophy. Sensory symptoms are usually prominent, and there is often tenderness along the nerve-trunks.

Syringomyelia.—In this affection the atrophy is accompanied by exaggerated reflexes, peculiar sensory disturbances, and trophic changes in the skin and joints.

Prognosis and Treatment.—The course of the disease is very slow and occasionally marked by remissions. Death may result from bulbar paralysis or intercurrent disease. Treatment is of no avail.

PRIMARY LATERAL SCLEROSIS

Definition.—A very rare disease, characterized by gradual loss of power, marked exaggeration of the deep reflexes, and a spastic condition of the muscles, without atrophy or sensory disturbances.

Etiology.—The etiology is obscure. The disease usually develops between the ages of twenty and forty, but there is a congenital and familial form which begins in the first few years of life. Both sexes are equally affected.

Pathology.—A primary degeneration of the lateral pyramidal tracts (terminations of the upper motor neurons) is the anatomic cause of the disease.

Symptoms.—Loss of power is usually the first symptom. This begins in the lower extremities and increases very slowly. The knee-jerk is exaggerated, and in most cases ankle-clonus can be elicited. When put in use, the muscles become stiff or spastic, and when the disease is fully developed, the gait is peculiar. In walking the knees are drawn together, the legs drag behind, and the toes catch the ground. The muscles do

not waste, sensory and trophic disturbances are absent, and the sphincters are rarely affected.

Diagnosis.—As an independent affection lateral sclerosis is rare, spastic paralysis of the legs usually being due to (1) *diseases of the brain involving both motor tracts*, such as infantile cerebral palsy, or (2) *diseases of the spinal cord dividing the lateral columns*, such as multiple sclerosis, syringomyelia, tumors of the cord, compression in Pott's disease, transverse myelitis, etc.

Prognosis.—The disease is incurable, but the course is extremely slow.

Treatment.—Rest, warm baths (90° F.), and massage are the most useful measures. If there be a suspicion of syphilis, antiluetic treatment should be instituted.

AMYOTROPHIC LATERAL SCLEROSIS

Definition.—A chronic disease, characterized anatomically by degeneration of the anterolateral columns and atrophy of the ganglionic cells in the anterior gray horns of the spinal cord, and clinically by progressive spasticity, paresis, muscular atrophy, and sooner or later symptoms of bulbar palsy. It is closely allied to spinal progressive muscular atrophy.

Pathology.—The chief lesion is a degeneration of the pyramidal tracts, with atrophy of the large cells in the ventral horns and of certain groups of cells in the medulla.

Symptoms.—These include wasting of the muscles, with loss of power, spastic contractions, exaggerated reflexes, and Babinski's sign. The upper extremities are usually first affected.

When the medulla is involved, symptoms of glossolabial-laryngeal paralysis appear (bulbar palsy). Sensation is not impaired, and the sphincters are rarely disturbed.

The muscular rigidity and exaggerated reflexes will distinguish it from pure *progressive muscular atrophy*, and the atrophy of the muscles from pure *lateral sclerosis*.

Prognosis and Treatment.—The course of the disease is exceedingly slow, but invariably progressive. The treatment is that of primary lateral sclerosis.

BULBAR PARALYSIS

(Glossolabiolaryngeal Paralysis)

Definition.—Paralysis of the lips, tongue, pharynx, and larynx from degeneration of the motor nuclei of the medulla oblongata.

Etiology.—An acute form is observed that results either from hemorrhage or from an acute poliomyelitis of the medulla. The chronic form (*progressive bulbar paralysis*) is essentially a chronic poliomyelitis of the bulb. It may occur as an independent disease, but more often it is a part of amyotrophic lateral sclerosis or progressive muscular atrophy.

Symptoms.—These include impairment of speech; inability to protrude the tongue; dribbling of saliva; difficult swallowing; choking spells from the entrance of food or mucus into the larynx; partial suppression of the voice with measured speaking; and a lack of facial expression. The paresis is attended by atrophy and fibrillary tremors.

In the rare disease known as *pseudobulbar paralysis*, which results from bilateral lesions in the motor cortex or internal capsule, there is usually some mental impairment and aphasia, and the paralysis is not accompanied by atrophy and fibrillary tremors. *Asthenic bulbar paralysis* (*myasthenia gravis*) is distinguished by the extreme fatigue after exertion, the ready exhaustibility of the muscles with faradism, the involvement of the ocular muscles (diplopia, ptosis), the absence of atrophy and the irregular course with remissions.

Prognosis.—The outlook is unfavorable. The acute variety is speedily fatal; the chronic form may last several years. Death may result from exhaustion, cardiac failure, or aspiration-pneumonia.

Treatment.—This is unsatisfactory. Massage and electricity may be tried. Strychnin has been recommended. The stomach-tube should be used when the patient is unable to swallow.

ACUTE ASCENDING PARALYSIS

(Landry's Disease)

Definition.—An acute disease of rare occurrence, characterized by motor paralysis beginning in the legs and rapidly spreading until it involves the muscles of respiration and deglutition.

Etiology.—The causes are unknown. The disease usually occurs between the ages of twenty and forty years, and in many cases is probably acute poliomyelitis of the adult or a form of acute myelitis.

Pathology.—In some cases degenerative changes have been found in the ganglion cells in the anterior horns of the spinal cord, in the peripheral nerves, or in both cells and nerves.

Symptoms.—Febrile symptoms usually usher in the attack. The paralysis begins in the legs and involves successively the trunk, upper extremities, and muscles of respiration and deglutition. The reflexes are abolished. The sphincters are retentive; sensation is usually normal, but there may be some paresthesia or even hypesthesia; the muscles are relaxed, but do not waste or yield the reactions of degeneration. In some instances the spleen and lymph nodes are swollen.

Diagnosis.—*Acute Myelitis.*—Anesthesia, muscular atrophy, reactions of degeneration, and early involvement of the sphincters will serve to distinguish myelitis from acute ascending paralysis.

Multiple neuritis may usually be distinguished from Landry's disease by the marked sensory disturbances (pain, local tenderness, anesthesia) in the former.

Prognosis.—The majority of cases terminate fatally in from a few days to two or three weeks. Occasionally the disease comes to a standstill and a slow recovery ensues.

Treatment.—The patient should be kept at rest, and wet cups applied to the spine. Extract of ergot (10 to 20 grains a day), belladonna, salicylates, mercury, and iodids are the remedies that have been recommended.

LOCOMOTOR ATAXIA

(Tabes Dorsalis ; Posterior Spinal Sclerosis)

Definition.—A progressive degeneration of the posterior roots and neurons, of syphilitic origin, characterized by incoördination, loss of deep reflexes, disturbances of sensation and nutrition, and various ocular phenomena.

Etiology.—The disease develops most frequently between the ages of 30 and 50 years and is much more common in men than in women. Overexertion, sexual excesses, exposure to cold, and alcoholic intemperance favor its occurrence, but the essential cause is syphilis.

Pathology.—The pia mater over the posterior columns is somewhat thickened and opaque. The posterior columns have a grayish color and are firm and shrunken.

Microscopic examination reveals degeneration and sclerosis of the dorsal-roots (usually in the lumbar region) and of the tracts and columns which these roots form in the spinal cord, especially the columns of Goll and Burdach, which transmit afferent impulses from the muscles, tendons and joints to the brain; hence impairment of coördination, sense of position, muscular tonus, and deep sensibility are conspicuous clinical features. In many cases degenerative changes are also found in the cranial nerves and nuclei, especially the optic and ocular nerves, and in the cerebral cortex (tabo-paralysis).

Symptoms.—The symptoms of the *early (pre-ataxic) stage* comprise paroxysms of sharp, shooting pains, usually in the legs, and frequently regarded as “rheumatic;” various forms of paresthesia, such as numbness and tingling of the feet, and a sense of constriction about the body (girdle pain); disturbances of the urinary and sexual functions, usually of a paretic

type; loss of the deep reflexes, especially of the knee-jerk (Westphal's sign); reflex iridoplegia (Argyll-Robertson pupil), the pupil showing no reaction to light, while still contracting in convergence and accommodation for near objects; the so-called visceral crises, the most familiar of which is the gastric crisis, characterized by paroxysms of intense epigastric pain, incessant vomiting, and the secretion of hyperacid gastric juice; isolated areas of hypesthesia or anesthesia, conforming to the root or segmental type, about the trunk and in the extremities; and an excess of lymphocytes, a marked albumin reaction and a positive Wassermann reaction in the cerebrospinal fluid.

In many cases accompanying the Argyll-Robertson pupil, there are other ocular disturbances, such as pronounced myosis, transient or persistent paresis of the external ocular muscles, manifested by squint, ptosis or double vision, and increasing dimness of vision, the result of optic atrophy.

One of the first obtrusive symptoms of the *ataxic stage* is a want of certainty and precision in the movements of the legs especially in the dark. If the patient stands erect, with his eyes closed and feet in juxtaposition, he sways and tends to fall (Romberg's symptom); or if the upper extremities are affected the ataxia becomes evident when he attempts to touch with his finger the tip of his nose. In the recumbent position, with his eye closed, he is unable to recognize the position in which his limbs are placed (loss of deep sensibility). In the course of time the gait becomes characteristic. The steps are awkward and jerky, the foot is raised high, projected forward and outward and brought down forcibly with a thud, the body is bent forward, and the eyes are riveted to the floor. Although there is no loss of muscular power in this stage, the muscles are abnormally flaccid and in consequence passive movements of unusual extent are possible (hypotonia). In many cases trophic phenomena also ensue, especially a perforating ulcer of the sole of the foot, abnormal brittleness of the bones, and the so-called arthropathies or Charcot's joints—

painless swellings of the large joints, with effusion, atrophy of the bones and cartilages, and ultimately luxation.

In from five to twenty years or more if the patient does not die of some intercurrent disease, the *paralytic stage* supervenes. This is characterized by progressive muscular weakness, incontinence of urine and feces, cystitis, bed-sores, and increasing marasmus.

In a certain proportion of cases the symptoms of paretic dementia (see p. 464) are associated with those of locomotor ataxia, giving rise to the condition known as *tabo-paralysis*.

Diagnosis.—*Cerebellar disease* may be differentiated by the form of the ataxia (reeling, with a tendency to fall in a certain direction), headache, vomiting, double optic neuritis, and absence of pupillary rigidity, Romberg's sign, Westphal's sign, shooting pains and spinal lymphocytosis. *Multiple neuritis* differs from tabes in the early appearance of motor paralysis, muscular atrophy, and tender nerve trunks, and in the absence of pupillary rigidity, lightning pains, girdle sensation, vesical disturbances and changes in the spinal fluid.

Prognosis.—Complete recovery probably never occurs. The duration ranges from three to twenty years. Death is usually the result of some intercurrent disease.

Treatment.—Rest is an important factor in the treatment. Erb advises that the patient should live as if he were an old man, quietly, regularly, and with no excitement. Mental fatigue should also be avoided. Sexual excesses are exceedingly injurious. The diet should be nutritious and easily digestible.

Massage affords a valuable means of securing the benefits of exercise without the expenditure of energy.

Systematic reëducation of coördinating movements, as originally recommended by Frenkel, has been found the most effective remedy for the ataxia. Even in advanced cases, in which there is marked disturbance of sensation, this method of treatment is not without benefit, and the improvement may last for years if the disease is stationary or only slowly progressive.

Tepid baths of 80°–85° F. are sometimes of service. They should be suspended, however, while the exercise treatment is being used. Arsphenamin, mercury and iodids should be given a thorough trial in all cases.

The Pains.—When the pains are severe, the most potent remedial measure is absolute rest in bed. Light touches of the actual cautery or sinapisms over the root of the nerve supplying the affected part often afford relief. Deep massage is sometimes of service. Mitchell has found the alternate application of ice and hot water useful. Flannel bandages applied firmly from the toes up to the middle third of the thigh sometimes do much good. A snugly fitting abdominal binder may also be used to lessen girdle pain. Electricity in the form of the faradic brush, static spark, or stable galvanic anode is worthy of a trial. Division of the posterior nerve-roots has recently been recommended.

The most generally useful anodynes are acetphenetidin and antipyrin. Nitroglycerin occasionally succeeds. In many cases recourse must be had to morphin, but its use should be deferred as long as possible.

Vesical weakness should receive the most careful attention. The bladder must be completely emptied—if need be, by catheterization. On the first appearance of cystitis the bladder should be washed out at frequent intervals with weak antiseptic solutions.

SUBACUTE COMBINED SCLEROSIS OF THE SPINAL CORD

(Posterolateral Sclerosis)

This is a degenerative process affecting simultaneously or consecutively the posterior and lateral columns of the spinal cord. It is most commonly the result of pernicious anemia, but other severe anemias, diabetes, pellagra, certain intoxications (ergot), and acute infections occasionally produce it. The symptoms represent a combination of those of tabes and spastic paraplegia, the most common being paresthesia, impairment of deep sensation (bathyanesthesia), especially of vibration and joint sensibility, slight incoördination in walking, slight paresis, and a positive

Babinski reflex. The knee-jerks are usually diminished or lost, but when the pyramidal tracts are more damaged than the posterior columns they may be increased.

FRIEDREICH'S ATAXIA

(Family or Hereditary Ataxia)

Definition.—A family disease, characterized anatomically by sclerosis of the posterior columns and crossed pyramidal tracts, and clinically by symptoms resembling those of locomotor ataxia. The *hereditary cerebellar ataxia of Marie*, in which there is degeneration of the spinocerebellar tracts or agenesis of the cerebellum itself, is apparently a variety of Friedreich's ataxia.

Symptoms.—The usual symptoms are: Ataxia of all purposeful movements; loss of the knee-jerks; irregular jerking movements of the hands; muscular weakness; nystagmus; a scanning speech; lateral spinal curvature, and deformity of the feet, generally talipes equinus with extension of the big toe.

It differs from *locomotor ataxia* in the absence of sharp pains, of sphincteric disturbances, Argyll-Robertson pupil, and crises, and by the occurrence of nystagmus, alterations of speech, and deformities of the feet and spine.

In *Marie's hereditary cerebellar ataxia* the symptoms are somewhat similar, but they usually appear after puberty, the knee-jerks are increased, atrophy of the optic nerves and paresis of the ocular muscles are common, and there is little tendency to scoliosis or club-foot.

Course.—The disease is progressive and incurable, but may last 20 or even 30 years.

DISSEMINATED CEREBROSPINAL SCLEROSIS

(Multiple Sclerosis; Insular Sclerosis)

Definition.—A chronic disease, characterized anatomically by patches of sclerosis of varying size scattered throughout the brain and spinal cord.

Etiology.—The causes are obscure. One of the acute infectious diseases or poisoning by one of the metals is not an infrequent antecedent. Trauma appears to have been a factor in some cases. Syphilis is without influence. The majority of cases occur in early adult life, between the fifteenth and thirty-fifth years.

Pathology.—Areas of sclerotic tissue, of various sizes and shapes, are found throughout the brain and spinal cord. The cranial nerves are often implicated.

Symptoms.—The most characteristic symptoms in well-developed cases are (1) increasing weakness in the lower extremities, with exaggerated tendon-reflexes and the Babinski sign; (2) a monotonous, scanning, or syllabic speech; (3) a tremor, especially marked in the hands, and appearing only during the performance of voluntary movements (intention tremor); (4) involuntary oscillation of the eye-balls, or nystagmus, upon fixing a near object or on looking to one side; (5) defective vision and optic atrophy, particularly pallor of the temporal sides of the optic discs; and (6) various subjective disturbances of sensation, such as headache, giddiness, and numbness or tingling in the limbs. In addition there may be diplopia, paresis of the ocular muscles, disturbances of micturition, mental impairment, hypesthesia, and epileptiform or apoplectiform seizures. The course is long, from two to twenty years, and remissions are common. Indeed, remission of the initial symptoms (paresthesia, ocular palsies, amblyopia, and weakness of one or more limbs) is somewhat characteristic.

Diagnosis.—*Paralysis Agitans.*—In this disease the face is mask-like and the attitude and gait are characteristic. The tremor decreases during effort and persists during repose; nystagmus and scanning speech are absent. *Hysteria* may simulate multiple sclerosis, but it does not present nystagmus, optic atrophy, or bladder disturbances.

Treatment.—The general treatment is the same as that of tabes dorsalis. Bromids and scopolamin are sometimes useful in reducing the tremors.

SYRINGOMYELIA

Definition.—A chronic affection of the spinal cord, characterized anatomically by the formation of a cavity in its substance, and clinically by atrophy of certain muscles, peculiar disturbances of sensation, and various trophic disorders.

Etiology.—It is much more common in males than in females. Eighty per cent. of the cases occur between the ages of ten and forty years. Acute infective diseases and traumatism are mentioned as causes.

Pathology.—The cavity is probably produced in the majority of cases by softening of overgrown neuroglia tissue (gliosis), which exists in consequence of some developmental anomaly. The cervical and upper dorsal regions are the usual seats of the lesion. The cavity lies in the gray matter, and may be in the position of the central canal or somewhat posterior to it. Secondary degenerations are frequently observed in the anterior or posterior horns or in the anterior or posterior columns.

Symptoms.—The disease usually attacks the upper extremities, the chief symptoms being wasting of the muscles; fibrillary tremors; loss of painful and thermic sensations, while tactile sensation is preserved or but slightly affected (*dissociation symptom*); lateral spinal curvature; and various trophic disturbances, such as arthropathies, fissures, ulcers, and gangrene. Such eye symptoms as nystagmus, inequality of pupils, and narrowing of the visual fields are frequently observed. In many instances symptoms of lateral sclerosis, posterior sclerosis, or bulbar disease are superadded.

The distinctive features of *Morvan's disease* (probably a form of syringomyelia) are tactile anesthesia and painless felons.

Diagnosis.—*Cervical pachymeningitis* is more painful, and the anesthesia includes tactile sensation. In *progressive muscular atrophy* and *amyotrophic lateral sclerosis* sensory symptoms are wanting. *Leprosy* may be recognized by loss of tactile sensation, discoloration of skin, nodular swellings, and presence of bacilli in the tissues.

Course and Treatment.—The course is slow, extending over many years, and the treatment is purely symptomatic.

CAISSON DISEASE

(Divers' Paralysis)

Definition.—A condition observed in divers and others subjected to increased atmospheric pressure, and characterized by motor and sensory paralysis and other nervous symptoms.

Etiology.—A pressure of more than two atmospheres is usually required to produce the paralysis, and the time elapsing before its appearance lessens as the pressure increases.

Pathology.—The symptoms are apparently due to congestion and foci of hemorrhages and softening in the cord caused by the liberation of gases (chiefly nitrogen) that have been absorbed during exposure to the high pressure.

Symptoms.—The condition may manifest itself immediately on reaching the surface or after the lapse of several hours. In the milder cases the symptoms consist of pains in the legs, abdominal cramps, paresthesias, and retention of urine. Occasionally, dyspnea and cough are also present. In the more severe forms there is paraplegia with retention or incontinence of urine and hypesthesia. Vertigo, staggering, tinnitus aurium, and neuralgic pains may also be observed. The most grave forms are marked by collapse, vomiting, cyanosis and unconsciousness. Unless the cerebral or circulatory disturbances are pronounced there is little danger to life, but paraplegia sometimes persists for months, and occasionally it is permanent.

Treatment.—As a preventive measure transition from high to low pressure should be accomplished gradually. Treatment consists in prompt recompression followed by gradual decompression. The after treatment of paralytic cases is that of myelitis.

PARALYSIS AGITANS

(Parkinson's Disease ; Shaking Palsy)

Definition.—A chronic progressive disease of the nervous system characterized by a gradually spreading tremor, muscular rigidity, and peculiar changes in posture, facial expression and gait.

Etiology.—The disease usually begins between the ages of 45 and 65 years and is comparatively rare before 35. Males are attacked more frequently than females. Psychic shock—fright, anxiety, grief, etc.—may be a provocative factor. In some instances the disease develops during or after epidemic encephalitis.

Pathology.—Recent studies seem to show that the essential lesion is atrophy or degeneration of the large motor cells (pallidal cells) of the lenticular nucleus of the striate body.

Symptoms.—In some cases the onset is abrupt, but more commonly the disease develops insidiously. A tremor appears in the hand, usually in the forefinger and thumb ("pill rolling" movement), and gradually spreads until it involves all the extremities and occasionally the neck and head. At first the tremor may be paroxysmal, but as the disease advances it becomes almost continuous. Excitement increases it, but it

is noteworthy that physical effort temporarily diminishes or checks it. The face becomes expressionless (mask-like), and the speech slow and measured. Later, muscular rigidity develops, and the head is bowed, the body bent forward, the arms flexed, the thumbs turned into the palms and grasped by the fingers, and the knees slightly bent. At this time the gait is characteristic. The steps are at first slow and shuffling, but they become progressively quicker and shorter until the patient is forced to run or come to a stop to prevent falling forward (*propulsion* or *festination*). Occasionally a tendency to fall backward (*retropulsion*) replaces festination. The rigidity and muscular weakness render all movements slow and stiff.

Intelligence is usually good. There is no anesthesia, but there are various manifestations of paresthesia, such as numbness and tingling and a sensation of heat. In some cases free perspiration is observed.

Diagnosis.—The tremor, rigidity, weakness, flexion of the body and members, lack of facial expression, and festination are the diagnostic features. In some cases the tremor is absent. Paralysis agitans must be distinguished from *disseminated sclerosis*. In the latter the tremor is absent when the patient is quiet, and is made worse by efforts to control it; cerebral symptoms are usually present; nystagmus is often noted; and the attitude and gait are entirely different from those of paralysis agitans.

Prognosis.—Recovery rarely, if ever, occurs, but the course of the disease is slow, often 20 or 30 years being reached before the final stage is reached.

Treatment.—Measures intended to improve the general nutrition are indicated. These include quiet living, adequate nourishment, abundant sleep and, in some cases, the use of tonics. Gentle exercise, active and passive, is beneficial. Electricity is harmful. Scopolamin in doses of $\frac{1}{200}$ to $\frac{1}{100}$ of a grain, two or three times a day, is sometimes of service in lessening the tremor. Bromids are also useful when there is much restlessness.

ACUTE CHOREA

(Sydenham's Chorea; Chorea Minor; St. Vitus' Dance)

Definition.—A relatively common disease, occurring chiefly in children, and characterized by involuntary muscular contractions of a disorderly nature and a pronounced tendency to endocarditis.

Etiology.—The large majority of cases occur in children between the ages of five and fifteen, though adults, especially women during or after pregnancy, are occasionally attacked. More females are affected than males. Heredity sometimes plays an important rôle. Fright or shock is not rarely an exciting factor. In about one-fifth of all cases there is an antecedent history of rheumatism between which disease and chorea there is a close relation. Chorea is most common in the spring months.

Pathology.—Minute hemorrhages and perivascular round-cell infiltration have sometimes been found in the basal ganglia and motor tracts. Recently, cases have been reported in which the changes were especially marked in the putamen and caudate nucleus of the corpus striatum and the cerebral cortex. In many cases a focus of infection is found in the tonsils.

Symptoms.—The first manifestations are usually restlessness and awkwardness in movement. The child cannot remain still, but is constantly raising its shoulders, jerking its head, twisting its fingers, or shuffling its feet. Frequently these symptoms develop so insidiously that the disease is not recognized, and the child is punished for being fidgety.

When the disease is fully established, the disorderly movements become more marked. They may be confined to one member or may involve the entire body. If the facial muscles are affected, the most grotesque expressions are produced; involvement of the arms may interfere with eating and dressing; if the legs suffer, the gait becomes jerking and stumbling; involvement of the larynx causes stammering, and spasm of the muscles of deglutition induces difficult swallowing and

choking spells. When the attention is directed to the movements, they invariably grow worse, but they diminish during repose and cease entirely during sleep. Sometimes, in addition to the involuntary movements, there is a distinct loss of power in the affected members. The general health is usually more or less impaired. The child is, as a rule, irritable and mentally dull.

Chorea Insaniens.—This form occurs chiefly in adults and most frequently in pregnancy. The movements are very violent, almost constant, and in many cases associated with delirium and fever. Death sometimes results from exhaustion.

Diagnosis.—The recognition of chorea is rarely attended with difficulty. In *habit spasm* or *tic* the movements are co-ordinated, purposeful, more localized and partly or completely under the patient's will.

Huntington's Chorea.—This disease is usually hereditary; it rarely develops before the age of thirty; it runs a chronic course; and it is characterized by slower and more incoördinate movements than occur in acute chorea, by progressive mental failure, and by a marked suicidal tendency.

Other conditions producing choreiform movements have been considered on page 444.

Complications.—Acute endocarditis occurs in from 25 to 30 per cent. of all cases. Pericarditis is occasionally seen. Rheumatic manifestations are sometimes present.

Prognosis.—In simple chorea recovery usually follows in the course of two or three months. Death from heart complications is a rare termination. Relapses are very common. *Chorea insaniens* sometimes terminates fatally through exhaustion.

Treatment.—Rest of body and mind is an essential element of the treatment. The child should be taken from school and placed under the most favorable hygienic conditions. Amusement in the open air when the weather is fine is to be recommended. In the more severe cases prolonged warm baths are often helpful. Arsenic has been extensively used, but it is

of doubtful value. Fowler's solution may be given in doses of two drops thrice daily after meals, and the dose gradually increased to 8 or 10 drops, unless untoward effects occur. Salicylates are worthy of trial when there are evidences of rheumatism. If the movements are very severe and interfere with sleep sedatives—bromids, barbital, chloral, scopolamin—must be used. Iron is indicated in anemic patients.

PROGRESSIVE LENTICULAR DEGENERATION

(Wilson's Disease)

This is a rare disease of adolescence, often familial, characterized anatomically by bilateral degeneration of the striate body (caudate nucleus, putamen, globus pallidus) and cirrhosis of the liver, and clinically by tremor, muscular rigidity, dysarthria, various psychic disturbances, and progressive weakness and emaciation. In the advanced stages contractures develop, dementia supervenes, and the patient becomes helpless and bedridden. The hepatic cirrhosis is usually latent. The disease is incurable, and may terminate within a few months, but it usually lasts several years.

HEADACHE

(Cephalalgia)

Headache of Organic Brain Disease.—This form is observed in meningitis, intracranial syphilis, cerebral tumor, abscess, softening, etc., and is usually distinguishable by its persistence and association with other evidences of organic cerebral disease, such as vertigo, vomiting, optic neuritis, and so-called focal symptoms. Headache due to intracranial syphilis is usually worse at night, and associated with somnolence and other signs of syphilis, such as a positive Wassermann reaction, etc.

Headache of Cerebral Hyperemia and Cerebral Anemia.—*Active cerebral congestion* usually results from prolonged mental work, fever, or exposure to the sun. Toxic and reflex headaches are often directly due to active cerebral congestion, but these will be discussed later.

Passive cerebral congestion may result from obstruction to the return of blood from the brain, as by a tumor of the neck or cardiac disease. It is also common in elderly persons from a relaxed condition of the vessels.

In cerebral congestion the headache is usually diffuse and of a throbbing character; the head is often hot and the face flushed.

Cerebral Anemia.—This is frequently dependent upon general anemia. It is also common in neurasthenia resulting from overwork, prolonged emotional excitement, excesses, etc. The pain is frequently vertical; it is not throbbing, but it is described as a sensation of weight or gnawing; the extremities are cold; the face and eye-grounds are pale; the mind is depressed; fainting spells are sometimes present, and lowering the head relieves the pain.

The exciting cause of cerebral hyperemia or anemia must be determined by the clinical history and a thorough examination of the various organs and blood.

Reflex Headache.—Headache is often due to *eye-strain*, and in obstinate cases a careful examination of the eyes should always be made. Headache of this origin is frequently a browache, and comes on in the afternoon, following the use of the eyes for near work. It is always induced or aggravated by prolonged use of the eyes.

Ovarian or uterine diseases often occasion a reflex headache. It is usually located at the vertex, and is relieved by pressure of the hand.

Gastric irritation is responsible for some headaches; the latter are relieved by vomiting, and are usually associated with other evidences of stomachic disorder.

Nasal catarrh may induce persistent headache, which is usually confined to the forehead, temples, or vertex, and is aggravated by exacerbations of the catarrh. The pain is associated with tenderness of the inner wall of the orbit, and is increased by irritating the nasal mucous membrane with a probe.

Toxemic Headache.—A persistent headache often results from renal disease, and is *uremic* in origin. It may be recognized by the high arterial tension and by the albumin and casts in the urine. A urinary analysis should be made in all cases of persistent headache.

Among other headaches of toxic origin may be mentioned the headaches due to alcoholism, to excessive use of tobacco, tea or coffee, to gout, to diabetes, to the acute infections.

Hysteric and Neurasthenic Headache.—In *hysteria* there is frequently a persistent headache, which grows worse at the menstrual periods, and which improves under pleasurable excitement. It may be diffuse, but frequently it is localized, and is described as resembling the effect that would be produced by a nail being driven into the head; hence it has been termed *clavus*.

The headache of *neurasthenia* is, as a rule, a dull pain or merely a sensation of fatigue. It is usually occipital but it may be diffuse. It is almost invariably aggravated by mental or physical effort and is associated with other neurasthenic phenomena, such as ready fatigue, backache and sleep disturbances.

Indurative Headache.—This comparatively rare form of headache, sometimes referred to as “rheumatic,” is frequently excited by chilling. It is increased by movements affecting the muscles of the head, and is associated with tenderness of the scalp and the presence of sensitive nodular swellings at points upon the skull corresponding to the insertion of the muscles.

Diagnosis.—Headache must be distinguished from *migraine*. In the latter the attacks are usually more distinctly periodic; the pain is often unilateral, and is frequently accompanied by vomiting, vasomotor disturbances, and subjective visual phenomena.

Headache in the region of the orbit may be mistaken for *acute glaucoma*, but in the latter condition the eye is inflamed; the cornea is hazy; the pupil is sluggish; vision is impaired;

and on palpation the affected eyeball is found to be harder than its fellow.

Treatment.—In the interval between the attacks careful search should be made for the cause, which, if possible, must be removed. In the reflex headache of eye-strain the adjustment of proper glasses is often all that is required. In the headache of gastric origin appropriate remedies should be directed to the stomach. In the headache of anemia a nutritious diet, with iron, arsenic, and other tonics, will be required. In headaches of uremic origin a milk diet with measures intended to increase the action of the skin, bowels, and kidneys will often afford considerable relief. Indurative headache is often favorably influenced by the wearing of a nightcap and the employment of gentle massage.

The Attack.—In headache dependent upon gastric acidity, after unloading the stomach with a non-irritating emetic, bromid with antacids will prove useful, thus:

R. Sodii bromidi..... ʒij
 Spiritus ammoniæ aromatici..... fʒij
 Aquæ..... q. s. ad fʒiij.—M.
 Sig.—A tablespoonful every hour or two.

In headache of acute cerebral congestion the feet should be soaked for ten or fifteen minutes in very hot water; an ice-bag placed on the head; and some sedative such as the following administered:

R. Acetphenetidini..... ʒj
 Sodii bromidi..... ʒss.—M.
 Fiant chartulæ No. xii.
 Sig.—One powder every two hours until relieved.

When the attack is very severe, aconite (3 or 4 drops) may be given every hour or two.

In cerebral anemia relief temporarily follows the use of antipyrin or acetphenetidini, especially in combination with caffeine, thus:

℞. Acetphenetidini..... ʒiss
 Caffeinæ citratæ..... gr. xxiv.—M.
 Fiant chartulæ No. xii.
 Sig.—One as required.

In indurative headache salicylic compounds are very useful; they may be combined with acetphenetidin or antipyrin:

℞. Acetphenetidini
 Salophen..... āā ʒiss.—M.
 Fiant chartulæ No. xii.
 Sig.—One every two or three hours.

Uremic headache will require rest, dietetic restrictions, and eliminative measures—catharsis, diuresis, diaphoresis, venesection, etc.

MIGRAINE

(Hemicrania; Megrim; Sick-headache)

Definition.—A neurosis characterized by periodic attacks of intense headache, often unilateral and frequently associated with visual, gastric, and vasomotor disturbances.

Etiology.—It is frequently hereditary. It is more common in women than in men. It usually develops in early life. Anemia, gastric disturbances, gout, eye-strain, menstrual disorders, overwork, and prolonged excitement predispose to it.

Symptoms.—Premonitory symptoms, such as restlessness, depression, and somnolence, are common. The attack is often ushered in with visual disturbances, such as flashes of light, colored spectra, dimness of vision, or hemianopsia. The pain is severe and usually limited to the temporofrontal region of one side, though it sometimes spreads until it involves the whole head. The patient is very sensitive to light and sound, and during the attack usually confines herself to a darkened room. Nausea and vomiting frequently supervene. In some cases vasomotor disturbances, such as pallor of the face and coldness of the extremities, or flushing with sweating, are also present.

Less frequent symptoms are numbness or tingling of one extremity, vertigo, tinnitus aurium, and transient motor weakness or aphasia. The duration of the attacks varies from a few hours to several days and of the intervals from a week to several months.

Diagnosis.—*Headache* is less distinctly paroxysmal, less localized, and unaccompanied, as a rule, by visual, gastric, or vasomotor disturbances. In *neuralgia* the pain is limited to the course of a nerve, tender spots are found, and vomiting and visual disturbances do not occur.

Prognosis.—The outlook for cure is not good, but much relief may be afforded, and usually the attacks become less severe or disappear entirely at the menopause or at the age of fifty years.

Treatment.—In the interval the treatment is that of neuralgia. *Cannabis indica* and arsenic are sometimes of value. Little recommends:

R̄. Sodii arsenatis..... gr. ij
 Extracti cannabis indicæ..... gr. vj
 Extracti belladonnæ..... gr. vj.—M
 Fiant pilulæ No. xxiv.
 SIG.—One pill twice daily.

The Attack.—The patient should be kept at rest in a quiet, darkened, well-ventilated room. Antipyrin, acetphenetidin, caffein, bromids, and salicylic compounds are the most useful remedies. They may often be combined with advantage, as in the following formulas:

R̄. Caffeinæ citratæ..... gr. xij
 Acetphenetidini..... ʒj
 Sodii bromidi..... ʒij.—M.

Fiant chartulæ No. xii.

SIG.—One powder every two hours.

R̄. Salophen..... ʒiss
 Acetphenetidini..... ʒj
 Caffeinæ citratæ..... gr. xij.—M.

Fiant chartulæ No. xii.

SIG.—One every two hours.

VERTIGO

(Dizziness; Giddiness; Swimming in the Head)

Definition.—A sense of unstable equilibrium in which the patient himself or surrounding objects appear to be in a state of rapid oscillation or rotation. It is a symptom of many conditions.

Etiology.—Vertigo may result from:

1. Disturbances of the cerebral circulation. The vertigo occurring in arteriosclerosis, arterial hypertension, chronic myocarditis, heart-block, valvular lesions of the heart and the severe anemias is included under this head.

2. Organic disease of the brain. Vertigo is especially common in lesions of the cerebellum, but it may also be present in lesions of the cerebrum.

3. Neuropathic conditions. Vertigo is not uncommon in hysteria, neurasthenia, traumatic neuroses, migraine, and epilepsy. In epilepsy it may precede, follow, or take the place of a convulsion.

4. Aural disturbances. Vertigo is most frequently observed in lesions of the labyrinth (Ménière's disease), but it may make its appearance as a result of disease of the middle ear.

5. Ocular disturbances. Ocular vertigo is usually dependent upon paresis of the ocular muscles, and is probably due to false projection of the retinal images. It is relieved by closing the eyes.

6. Toxemic conditions. Vertigo is sometimes observed in indigestion, gout, uremia, diabetes, acute infections, and in poisoning by tobacco, alcohol, lead, and many other substances.

7. Mechanical causes. The vertigo experienced in seasickness, swinging, whirling, etc., is probably dependent upon violent excitation of the semicircular canals, produced by the rapid movements of the body.

Diagnosis and Treatment.—The cause of vertigo must be determined by a careful study of the associated symptoms and

a thorough examination of the heart, blood-vessels, eyes, ears, urine, blood, etc. Bárány's caloric test (syringing the ear with hot or cold water) and rotation tests are of great value in localizing the lesions responsible for vertigo. The absence of an obvious cause, the short duration of the attacks, the occurrence of actual unconsciousness and after-effects (malaise, confusion, etc.) will usually serve to differentiate *minor epilepsy* from vertigo. The treatment of vertigo is, of course, the treatment of the affection of which it is a symptom.

MÉNIÈRE'S DISEASE

The term Ménière's disease is applied to a symptom-complex characterized by deafness, tinnitus aurium, and paroxysms of intense vertigo. The paroxysms, which may occur daily or at intervals of weeks, may culminate in vomiting or even in syncope. The condition usually continues through life, but occasionally recovery ensues when the loss of hearing on the affected side becomes absolute. The lesion is apparently in the labyrinth and may be degeneration of the nerve-ends or hemorrhage. In some instances it follows disease of the middle ear. It should be borne in mind that tumors of the cerebello-pontile angle may excite similar symptoms.

Treatment.—The middle ear should be carefully examined and any existing disease treated. Counterirritation behind the ear (actual cautery or small blisters) may be tried. Bromids in full doses, gelsemium, and suprarenal extract (5 grains thrice daily) may be of service. Quinin in ascending doses (Charcot) and pilocarpin, $\frac{1}{6}$ grain once a day hypodermically (Politzer), have also been recommended.

EPILEPSY

Definition.—A chronic disease of the nervous system, characterized by periodic attacks of unconsciousness or disturbed consciousness, with or without convulsions.

Etiology.—A neuropathic inheritance is the most important etiologic factor. Chronic alcoholism and syphilis in the parents may also act as predisposing causes. Not rarely

the disease follows an injury of the brain or one of the infectious fevers. The reflex convulsions of infancy, if frequently repeated, may serve as an exciting cause in those predisposed. Essential epilepsy usually begins before the age of puberty, and rarely develops after the thirtieth year. In epileptic subjects overwork, digestive disturbance, excitement, or menstruation may be the immediate cause of an attack.

Pathology.—No constant lesions are revealed at autopsy. Morphologic anomalies in the cerebral convolutions and in the organism as a whole indicative of defective development are, however, frequently noted. The disease depends apparently upon an excessive irritability of the motor cells of the cerebral cortex.

Symptoms.—*Major Epilepsy (Grand Mal).*—In about one-half of the cases the convulsion is preceded by a warning symptom, or aura. This may consist of a sensation of numbness beginning in one extremity, muscular twitchings, flashes of light, a peculiar odor, or a certain emotion. After the aura the patient emits a shrill cry, becomes pale, and falls to the floor unconscious and convulsed. At first the body is thrown into a tonic spasm, with the head rotated and retracted, the limbs forcibly extended, and the thumbs turned into the palms and firmly clenched by the fingers. The respiration ceases and cyanosis follows. In a few seconds the tonic spasm gives way to a clonic spasm in which the movements are jerky, rapid, and often violent. During this stage the breathing is stertorous, the tongue is often bitten, frothy saliva, frequently blood-streaked, exudes from the mouth, and the urine is discharged. The whole attack usually lasts two or three minutes. At the close, the patient may remain in coma for a variable time or he may rapidly recover consciousness, and afterward complain of mental confusion, headache and muscular soreness. Occasionally the attack is followed by a trance-like state, by epileptic automatism (automatic performance of purposive acts), by epileptic delirium, perhaps attended by violent assaults, or by transient paralysis. In some instances

automatism or delirium (an epileptic equivalent) replaces a convulsion.

Petit Mal.—In this form the seizure usually consists of momentary unconsciousness, sometimes accompanied with pallor, or more rarely, slight starting of the head or limbs. The patient suddenly stops in the midst of his work or conversation, remains still for a few seconds, and then reverts to his former activity, as if nothing had happened. Occasionally, there is only obscuration of consciousness and the attack is represented by a dreamy, “twilight” state, a sudden sensation of fear or horror, or an hallucination. *Petit mal* may be a forerunner of *grand mal* or may alternate with it.

Between the extremes of *grand mal* and *petit mal* attacks of every degree of severity are observed. The attacks may occur daily, several times a day, or at intervals of weeks months, or years.

The term *status epilepticus* is applied to a condition in which convulsions follow one another in rapid succession without a return of consciousness.

No other symptoms may be present in epilepsy except those pertaining to the paroxysms, but in cases in which the latter frequently recur mental deterioration, as a rule, gradually supervenes.

Diagnosis.—In *organic disease of the brain* intercurrent symptoms are usually present, and not rarely the convulsions are limited to one member or habitually begin in one member and then become general (Jacksonian epilepsy). An ophthalmoscopic examination often aids in the diagnosis. Convulsions occurring for the first time in persons over thirty years of age are usually the result of organic brain disease or of an intoxication (uremia, alcohol, etc.).

Prognosis.—Arrest of the disease is exceptional, but marked amelioration is frequently observed under appropriate treatment. According to Starr about 10 per cent. of epileptics eventually become insane. Death rarely occurs in a convulsion although the status epilepticus may kill by exhaustion.

Treatment.—Hygienic treatment is of the utmost importance. Moderate exercise, both mental and physical, is beneficial. Idleness and seclusion have a baneful effect. Home training must be carried on with the greatest care, much tact and firmness being required to prevent loss of self-control. Children who are particularly irascible are often much better trained in a special institution. As a rule, a diet that is for the most part vegetable will be found to be best adapted to the patient's condition, but when the disease is associated with lowered vitality, a fair amount of animal food should be permitted. Overloading the stomach is a potent factor in precipitating attacks. The bowels must be regulated by diet, and, if necessary, by mild aperients. Liberal water-drinking, frequent bathing, followed by friction of the skin, light exercise in the open air, and other measures that favor elimination are to be recommended. General tonics, such as iron, arsenic, and cod-liver oil, are sometimes required to combat anemia and malnutrition.

Although very few cases of epilepsy are purely reflex, local irritation—phimosis, adherent prepuce, worms, a foreign body in the nose or ear, and painful cicatrices—should be carefully sought for and, if found, removed.

The most reliable drugs are the bromids and phenobarbital (luminal). One or two drams of a combination of the bromids of sodium, potassium, and ammonium may be given daily. Strontium bromid is often efficacious, and it is less depressing than the other bromids. The tendency to acne may be lessened by the addition of a drop or two of Fowler's solution to each dose of bromid. A small amount of antipyrin sometimes lessens the amount of the bromid required to check the convulsions but it must not be used over long periods.

℞. Potassii bromidi
 Ammonii bromidi..... āā ʒiij
 Liquoris potassii arsenitis..... fʒss
 Antipyrinæ..... ʒj
 Aquæ menthæ piperitæ..... q. s. ad fʒvj.—M.

Sig.—A tabiespoonful in water night and morning.

In nocturnal epilepsy chlorbutanol (chloretone) (5 grains) is often a useful adjuvant to the bromids. In some cases small doses of thyroid extract ($\frac{1}{2}$ –1 grain) increase the efficiency of the bromids.

Phenobarbital in doses of 1 to 2 grains at bedtime, or $\frac{1}{2}$ grain thrice daily, is often superior to the bromids.

Operation offers some hope of relief in certain cases of focal epilepsy, although it has to its credit less than 4 per cent. of recoveries.

The Attack.—If there is a well-marked aura, the attack may be averted at times by the inhalation of amyl nitrite. Patients may provide themselves with this drug in the form of pearls that may be crushed in the handkerchief. If a sensory aura is felt in a limb, the part may be firmly grasped or encircled with a light ligature. During the attack the head should be slightly raised, the clothes loosened, and a piece of cork, wrapped in a handkerchief, pushed between the teeth. In the status epilepticus the most reliable measures are inhalations of chloroform, ether, or amyl nitrite, hypodermic injections of scopolamin ($\frac{1}{100}$ grain) or of morphin ($\frac{1}{4}$ grain), enemas of chloral (20 to 30 grains), and the hot bath.

HYSTERIA

Definition.—A psychoneurosis characterized by an abnormal susceptibility to suggestion and a lack of self-control, and manifested by a train of symptoms of the most varied character.

Etiology.—Females are especially predisposed, although it occasionally develops in males. The disease is most common in early adult life. Heredity is an important etiologic factor, the disease frequently being transmitted through hysteric, epileptic, degenerate, or insane parentage. Faulty home-training and education also do much to foster its development. Traumatism, prolonged emotional excitement, such as worry, anxiety, disappointment, grief, and all causes that reduce vitality serve to excite it in susceptible individuals.

Freud ascribes hysteria to a mental conflict (suppression of memories) which has arisen as a result of painful experiences (often sexual events) in early childhood, and would make the vivid recollection of these experiences, under insistent questioning, and a complete verbal confession of them to the physician, an essential element of treatment.

Symptoms.—The various manifestations may be described under three heads: (1) Motor, (2) sensory, and (3) psychic.

Motor Phenomena.—Paralysis not infrequently results from hysteria; it may take the form of a hemiplegia, paraplegia, or monoplegia, although the first is by far the most common. The paralysis is usually paroxysmal, and is frequently associated with contractures and anesthesia. The affected muscles do not waste.

Local paralysis is also common; thus there may be aphonia from paralysis of the vocal cords; dysphagia, from paralysis of the esophagus; and incontinence of urine, from paralysis of the bladder.

Convulsive seizures are common manifestations of hysteria, and may closely simulate the paroxysms of true epilepsy, but there is no aura; the patient usually falls in a comfortable place; consciousness is only apparently lost; the tongue is rarely bitten; the eyes are partially closed; the face is expressive of some emotion; screaming or sobbing is of frequent occurrence; the movements are usually tonic, or if clonic, of wide range and purposive; the seizures are of long duration, and may be continued for several hours, and there is no involuntary micturition.

The spasms may be local; thus there may be retention of urine from spasm of the bladder; hiccup, from spasm of the diaphragm; dysphagia, from spasm of the esophagus; or a "phantom tumor," from spasm of the abdominal muscles.

Among other motor phenomena may be mentioned obstinate tremors, choreiform movements, and muscular contractures.

Sensory Phenomena.—There may be a complete loss of sensation in certain parts, as of one side of the body. Anes-

thesia without other nervous phenomena is usually hysteric. In some cases tactile sensation is preserved and there is a loss only of thermic or painful sensations. The anesthetic part is sometimes unusually pale, and when pricked with a needle, may fail to bleed.

The special senses may be involved; thus there may be contraction of the field of vision, complete blindness, loss of smell, loss of taste, or loss of hearing. These palsies are usually transient, and sometimes alternate with one another.

Instead of anesthesia, there may be hyperesthesia or pain. An intensely painful and tender condition of the abdomen may be mistaken for peritonitis. A localized pain in the head, described as resembling the effect of a nail being driven into it, is termed hysteric *clavus*. The joints occasionally become swollen and tender, resembling arthritis (neuromimesis).

Psychic Phenomena.—As a rule, the most conspicuous psychic phenomena are an abnormal susceptibility to suggestion, introspectiveness, incapacity to exercise control over emotions and actions, and a morbid craving for sympathy and attention. Hysterical patients are excitable, highly mercurial, and easily moved to laughter or tears. Occasionally delirium, ecstasy, catalepsy, trance or somnambulism makes its appearance.

Treatment.—This must be directed both to the mind and the body, especially to the former. To be successful, the physician must be able to inspire absolute confidence and faith in the mind of the patient. She must be impressed repeatedly with the fact that her condition is a curable one, and that with her thorough coöperation restoration to health will certainly follow. To intimate that her symptoms are feigned or are wholly within her control is a grave error. In many cases no method of treatment proves successful until the patient has been removed from her customary surroundings and separated from her sympathetic relatives and friends.

Suggestion is employed consciously or unconsciously in the treatment of hysteria by every successful physician.

Without it many of the remedies recognized as efficacious become wholly impotent. Complete hypnotism, however, is by no means so generally useful as continuous suggestion, and, moreover, in the event of failure, seems capable of still further lowering the will-power and of increasing the emotional excitability.

The physical condition of the patient must not be neglected. General measures, such as hydrotherapy, systematic exercise in the open air, massage, and electricity, are valuable aids to recovery. In grave cases the treatment associated with the name of S. Weir Mitchell often yields admirable results. It consists in isolation from sympathizing friends and relatives; abundant feeding, especially with milk; and complete rest of body and mind, with passive exercise obtained by massage and electricity.

Except to meet underlying conditions and to combat special symptoms, drugs are of little value. Iron and arsenic are useful when there is anemia. Antispasmodics, such as valerian, sumbul, asafetida, and camphor, are serviceable in allaying abnormal nervous irritability.

Such combinations as the following may prove useful:

℞. Arseni trioxidi..... gr. ss
 Ferri pyrophosphatis
 Extracti sumbul..... āā gr. xx
 Asafoetidæ..... gr. xl.—M.

Fiant pilulæ No. xx.

SIG.—One three or four times a day.

℞. Quininæ valeratis
 Zinci valeratis
 Ferri valeratis..... āā gr. xxiv.—M.

Fiant pilulæ No. xxiv.

SIG.—One pill thrice daily.

Occasionally more powerful sedatives, such as the bromids, acetphenetidîn, barbital, and chloralamid, may be required, but the continuous use of such remedies is always to be condemned. Such drugs as morphin, alcohol and chloral are distinctly dangerous.

When hysteria is complicated by local disease, special treatment will be required, but no operation should ever be performed for the relief of nervous symptoms unless there exists an actual organic lesion.

Convulsions.—Isolation of the patient is imperative. Firm pressure over the ovarian region is sometimes successful. The affusion of cold water over the face is useful. Inhalations of amyl nitrite, or even of chloroform, may be employed if necessary. Strong faradic currents applied to the spine are occasionally efficacious.

Anesthesia is best treated by electricity, especially by the faradic brush. Static electricity, owing to the profound mental impression which it produces, is also useful. *Hyperesthesia* and *pain* often yield to the continuous or interrupted galvanic current. In *paralysis* the patient should be instructed how to regain, by long-continued practice, the use of the affected part. Swedish movements, massage, and faradization are useful adjuvants. In *aphonia* the faradic current, one electrode over the larynx and the other over some indifferent point, is sometimes effective. In *contractures* the most useful measures are massage, passive movements, and faradization.

NEURASTHENIA

Definition.—Neurasthenia is a neurosis affecting various organs and functions and characterized by ready and persistent exhaustibility and increased nervous irritability.

Etiology.—*Secondary neurasthenia*, caused by organic disease, such as tuberculosis, arteriosclerosis, diabetes, syphilis, etc. is very common. *Primary neurasthenia* is relatively uncommon. It usually develops in early adult life, and rarely appears for the first time after 40. An inherited neuropathic taint is an important predisposing factor, and mental or physical overwork, prolonged emotional strain, the shock of an injury (traumatic neurasthenia), sexual or alcoholic excess, and exhausting illnesses are common determining causes.

Symptoms.—*Psychic Disturbances.*—These include diminished capacity for continued mental work, irritability of temper, introspectiveness, emotional depression, and feebleness of volition. The quality of the mind is not impaired.

Sensory Disturbances.—Dull headache and backache are usually present. The latter is often accompanied by marked sensitiveness of the entire spine or of certain vertebræ. Vague pains in various parts of the body are common, but anesthesia is never observed.

Motor Disturbances.—Muscular fatigue after slight effort is the most constant symptom. A fine tremor may also be present. Actual paralysis never occurs.

Somatic Disturbances.—These are varied and include the symptoms of nervous dyspepsia (see p. 49), palpitation, paroxysms of pseudoangina pectoris, signs of vasomotor instability (hot flushes, coldness of the extremities, excessive throbbing of the abdominal aorta, etc.), and various sexual derangements, such as impotence, nocturnal emissions, and dysmenorrhea.

Diagnosis.—The diagnosis is rarely difficult. Before relegating a case to this class, care must be taken to exclude *organic disease of the various organs* and such disorders as hyperthyroidism, diabetes, arterial hypertension, cerebrospinal syphilis, multiple sclerosis, general paresis, chlorosis, etc.

Hysteria.—This affection may be distinguished by the abrupt onset, the intermittent character of the symptoms, and such stigmata as paralysis, anesthesia, contractures, emotional outbreaks, and convulsions.

In pure *psychasthenia* there is no ready exhaustion and the mental condition is characteristic, its chief manifestations being timidity, lack of decision, apprehensiveness, extreme weakness of will and a sense of utter insufficiency. Phobias are also common.

In *hypochondriasis* there are no fatigue symptoms, the essential feature being a dominating, but unfounded, conviction of illness.

Prognosis.—If the cause can be removed and the patient controlled, the prognosis is favorable.

Treatment.—The treatment is largely hygienic and dietetic, and must vary considerably in different cases. If there has been inactivity, regulated physical exercise will be of great value; on the other hand, the weak and anemic will require rest. In the latter case the plan of treatment introduced by S. Weir Mitchell, and known as the “rest-cure,” often gives brilliant results. In all cases careful attention must be given to the diet, bathing, and clothing, and the patient assured that he is suffering from no incurable disease. Frequent bathing with salt water, followed by friction of the skin, will often add to the general vigor. Tobacco and alcohol must be interdicted, and tea and coffee used very sparingly. Tonics, such as iron, arsenic, quinin, strychnin, and phosphorus, are often indicated.

OCCUPATION NEUROSES

An occupation neurosis is a form of muscular spasm induced by the frequent or prolonged execution of certain coördinated movements, and occurring only in the performance of work requiring those particular movements. The condition occurs chiefly in neurotic individuals as the result of overwork, and is observed most frequently in writers (*writer's cramp* or *palsy*), telegraph operators, pianists, typewriters, and seamstresses. It is probably due to exhaustion of the cerebral centers concerned in the execution of the movements which are affected by the spasm. In writer's cramp, which may be taken as a type of these disorders, the patient experiences a sense of fatigue or a dull ache in the wrist and hand and a tonic or clonic spasm of the muscles whenever he attempts to write. In some cases muscular weakness and tremors are also present. Neuritis can be excluded by the ability to execute all other movements involving the same muscles, and by the absence of tenderness along the course of the nerve and of muscular atrophy. The prognosis is fairly good for recovery,

if a sufficient period of rest can be enforced. Relapses, however, are very common. Therapeutic measures comprise the absolute cessation of the activities that have led to the neurosis, massage, outdoor exercise, hydrotherapy, galvanism or the static breeze, and the administration of general tonics.

LOCALIZED NEURITIS

Etiology.—Inflammation of a nerve may arise from (1) trauma, as blows, stretching, compression, or wounds; (2) exposure to cold; (3) the extension of inflammation from adjacent structures; (4) infectious diseases (5) poisons, such as alcohol or lead.

Pathology.—The sheath, interstitial connective tissue, or fibers may be independently affected, but, as a rule, all parts of the nerve are involved. In *acute cases* the nerve is red and swollen, and microscopic examination reveals cellular infiltration, with more or less fatty and granular degeneration of the fibers. In *chronic neuritis* the nerve-trunk is gray, shrivelled, and hard, and microscopic examination shows an overgrowth of connective tissue and granular degeneration of fibers.

Symptoms of Acute Neuritis.—There are usually three sets of phenomena—sensory, motor, and trophic—although the last may be absent in mild forms of the disease.

Sensory Symptoms.—There is severe pain following the course of the affected nerve, which is tender to the touch. The pain is often associated with various paresthesias, such as burning, numbness, tingling, etc. The part is at first hyperesthetic, but later it is more or less anesthetic.

Motor Symptoms.—Muscular power is impaired; there may be fibrillary tremors; the reflexes are diminished or lost.

Trophic Symptoms.—An eruption of herpes sometimes follows the affected nerves. The skin may become glossy and the nails lusterless and brittle. In advanced cases the muscles undergo atrophy and yield the reactions of degeneration.

Chronic neuritis is characterized by pain, hypesthesia, paresis, atrophy and contracture of the muscles, reactions

of degeneration, "glossy skin," and thickening and brittleness of the nails.

Diagnosis.—Neuritis may be mistaken for *neuralgia*, but in the latter the pain is paroxysmal, the tenderness is limited to certain points, and paresthesia, anesthesia, paresis, and changes in the electrocontractility are absent.

Prognosis.—In acute cases the prognosis is usually favorable; the duration is from a few days to several weeks. In chronic neuritis, after the development of marked trophic changes, the prognosis should be guarded.

Treatment.—The cause should be ascertained, and, if possible, removed. The part should be put at rest. For the pain, sedative lotions (saturated solution of Epsom salt on several thicknesses of gauze), warm fomentations, or small blisters may be applied to the affected parts, and a salicylate or acetphenetidin, with or without codein, administered. Such a combination as the following is often efficacious:

R _x .	Acetphenetidini.....	5j
	Acidi acetylsalicylici.....	5iss
	Codeinæ sulphatis.....	gr. iiij

Ft. Chart No. xii.

SIG.—One every three or four hours.

After the symptoms of irritation have completely subsided massage and electric treatment should be employed to promote nutrition in the paralyzed muscles.

MULTIPLE NEURITIS

Definition.—Inflammation of several nerve-trunks resulting from a general cause, and characterized by pain, paresthesia, anesthesia, paresis, and muscular atrophy.

Etiology.—The disease may result from (1) specific infections, such as diphtheria, influenza, measles, septicemia, rheumatism, etc.; (2) certain poisons derived from without, such as alcohol, lead, arsenic, carbon monoxid, sulphonal, etc.; (3) certain auto-intoxications, such as occur in gout and diabetes;

(4) malnutrition, as in advanced arteriosclerosis. In the Orient multiple neuritis occurs endemically (*beriberi*) as a result of a defective diet (see p. 436).

Symptoms.—The **acute form** is characterized by chilliness, moderate fever (102° – 103° F.), pains in the head and back, anorexia, constipation, and the following local phenomena: pain, numbness, and tingling in the affected limbs, loss of power, especially in the legs and extensor muscles of the wrists, tenderness over the nerve-trunks, abolition of reflexes, and, later, hypesthesia or anesthesia.

Death may occur within one or two weeks from cardiac or respiratory paralysis. Usually, however, recovery follows in from a few weeks to several months.

Chronic Form.—*Sensory symptoms* are usually prominent. Numbness, tingling, hyperesthesia, and intermittent pains appear early and are followed by hypesthesia or anesthesia, especially of the legs and hands. The nerve-trunks are sensitive. In certain forms of neuritis, as that resulting from diphtheria or from lead poisoning, sensory disturbances are frequently absent.

Motor Symptoms.—Weakness of the legs and forearms develops rapidly. The typical paresis is foot-drop and wrist-drop. The sphincters are not affected. The knee-jerks are lost. As a rule the paralyzed muscles do not respond to the faradic current, but yield the reactions of degeneration with the galvanic current. If the patient is able to walk, his gait is characteristic. To avoid dragging his toes, he raises the foot high, throws it suddenly forward, and brings it down flat on the floor, as if walking over obstacles (steppage gait).

Vasomotor and Trophic Disturbances.—The paralyzed muscles are flabby and soon waste. Edema of the feet and hands, and local sweating are often seen. Changes in the nails are common. Bedsores do not appear.

Mental Symptoms.—Forgetfulness, confusion, disorientation, and narration of pseudoreminiscences (Korsakow's syndrome) are not rare, especially in the alcoholic form.

Diagnosis.—*Locomotor Ataxia.*—The absence of the lightning-pains, girdle sensation, Argyll-Robertson pupil, sphincteric disturbances, and characteristic changes in the spinal fluid, and the presence of paralysis, wasting, and neural tenderness will serve to distinguish multiple neuritis from locomotor ataxia.

Acute myelitis may be distinguished by the absence of severe pains and of tenderness over the nerves, the presence of complete anesthesia and of sphincteric disturbances, and by the tendency to bedsores.

Prognosis and Treatment.—As a rule, the prognosis is good, if the exciting cause can be removed and the general health of the patient is not seriously impaired. The treatment is similar to that of localized neuritis.

FACIAL PARALYSIS

Etiology.—Paralysis of one side of the face may result: (1) From a tumor, clot, or abscess involving the facial center in the cortex of the brain or the nucleus of the facial nerve; (2) from the pressure of inflammatory exudate on the nerve-trunk between the brain and the skull; (3) from paralysis of the nerve within the petrous portion of the temporal bone, excited by a fracture or by an extension of inflammation of the middle ear; (4) from inflammation of the peripheral filaments, excited by exposure, injury, diabetes, or one of the infectious fevers.

Symptoms.—The side affected is expressionless; the natural lines are obliterated; the angle of the mouth droops; the eye cannot be closed; tears flow over the cheek; and speech is affected from an inability to pronounce the labials. When the patient attempts to laugh or whistle, the absence of movement on the affected side becomes still more conspicuous. In peripheral neuritis the reflexes are abolished; and when the nerve is involved in the temporal bone, there may be a loss of taste in the anterior part of the tongue.

Diagnosis.—When the lesion is supranuclear the upper muscles of the face (orbicularis palpebrarum and frontalis) usually escape, voluntary movements are more impaired than emotional movements, electric reactions are normal, and there is usually hemiplegia.

When the lesion is nuclear or infranuclear (*Bell's palsy*) all the muscles of one side of the face, including those of the forehead and eye, are involved, both emotional and voluntary movements are lost, and the electric reactions are altered in character. In nuclear lesions other cranial nerves are usually involved with the facial. In pontine lesions there is often paralysis of the limbs on the side opposite to the facial palsy (crossed paralysis). When the nerve is involved within the Fallopian canal there is frequently loss of taste in the anterior part of the tongue on the paralyzed side.

Prognosis.—The prognosis will vary with the cause. It should be guardedly favorable when the paralysis is due to peripheral neuritis.

Treatment.—The cause should be ascertained, and, if possible, removed. In paralysis of central origin little can be done except in syphilitic cases. In middle-ear disease remedies should be directed to that origin. When paralysis results from inflammation of the peripheral filaments of the facial nerve, blisters should be applied near the stylomastoid foramen. Later, a course of iodid of potassium will be useful, and restoration of power may be materially assisted by massage and electricity.

SCIATICA

Definition.—Pain in the course of the sciatic nerve.

Etiology.—The pain is usually referred to the nerve, the lesion itself being in some closely related structure, most frequently the lumbosacral spine, or the sacroiliac or hip joint and consisting of infective arthritis, or more rarely tuberculosis or carcinoma. In other cases the cause is inflammation or carcinoma of the prostate. In some instances pressure

on the plexus within the pelvis by inflammatory exudate, a tumor or hardened feces is a factor. Much less frequently sciatica is the result of a true neuritis arising from diabetes, syphilis, gout or alcoholism. Individual attacks are often traceable to chilling of the body or to direct pressure by a hard object.

Symptoms.—The disease may begin abruptly or gradually, and is characterized by a sharp shooting pain running down the back of the thigh. Movement of the limb intensifies the suffering. The pain may be uniformly distributed along the course of the nerve, but not infrequently there are certain spots where it is more intense. Subjective sensations, such as tingling and numbness, are often noted. The nerve may be extremely sensitive to touch. The symptoms grow worse at night and on the approach of stormy weather. In long-standing cases there may be much wasting of the muscles.

Prognosis.—Recovery follows in the majority of cases if treatment is instituted early and is persistently carried out. Relapses, however, are common, and in some cases the pain becomes more or less continuous.

Treatment.—The first indication is to remove the cause. In acute cases rest in bed is essential. In severe cases the limb should be immobilized by means of salt bags or a long straight splint. Free evacuation of the bowels should be secured in order to deplete the pelvic veins. Irrespective of the cause such drugs as the salicylates, acetphenetidin, cinchophen (atophan), in full doses, are often effective in relieving the pain. Morphin is sometimes necessary. High-frequency electric currents may be of service. Counterirritation by dry cups or small blisters along the course of the nerve is also useful. Very intense pain sometimes yields to injections of guaiacol (2–3 minims) made deeply near the nerve. Arthritic cases will require removal of the primary infective focus and appropriate orthopedic measures. When no organic abnormalities outside of the sciatic nerve are discoverable perineural or epidural injections of normal salt solution are worthy of trial.

NEURALGIA

Definition.—Paroxysmal pain occurring along the peripheral ramifications of a nerve and caused by pathogenic irritation affecting the nerve at some point or other in its course.

Etiology.—It is a disease of adults. Women are more frequently affected than men. Heredity is an important etiologic factor. The disease is sometimes an expression of anemia. It may result from the action of some toxic agent in the blood; thus it is common in malaria, diabetes, gout, and chronic lead-poisoning. It may be caused by reflex irritation; thus a trifacial neuralgia may depend on caries of the teeth or eye-strain. In some cases neuralgia results from organic disease of the ganglion of the nerve or the central sensory neuron; thus refractory trifacial neuralgia may be dependent upon degeneration or tumor of the Gasserian ganglion.

Exposure to cold and wet frequently acts as an exciting cause in susceptible persons.

Symptoms.—The chief symptom is intense pain of a sharp, stabbing character. The area supplied by the affected nerve is usually hyperesthetic, and palpation may detect spots of extreme tenderness where the nerve makes its exit through a bony canal or fibrous sheath—*puncta dolorosa* of Valleix. In some cases the pain is attended with reflex spasm of the muscles. Inspection of the part usually reveals nothing abnormal, but in some cases slight swelling is observed. Occasionally herpes precedes or follows an attack.

The attack lasts from a few minutes to many hours, and its subsidence may be marked by the passage of a large amount of pale urine. The interval between the paroxysms varies in different cases; it is frequently several weeks or months. It is noteworthy that the attacks often recur at regular intervals.

Trifacial Neuralgia (Tic Douloureux; Prosopalgia).—In this variety the pain involves one or more branches of the trifacial nerve. The tender points correspond to the supra-orbital, infra-orbital, and mental foramina. Reflex spasms of the

masticatory muscles are not infrequently observed. In long-standing cases the hair on the affected side may become coarse and bleached.

Intercostal Neuralgia.—In this variety the pain follows the course of the intercostal nerves. It is frequently associated with an eruption of herpes zoster. Spots of tenderness may be detected near the vertebral column, in the mid axilla, and near the sternum. The possible dependence of intercostal neuralgia upon spinal caries or thoracic aneurysm must not be forgotten.

Occipital neuralgia involves the upper cervical nerves. A spot of tenderness may be discovered midway between the mastoid process and the upper cervical vertebræ. This form of neuralgia may also be an expression of spinal caries.

Diagnosis.—*Neuritis.*—The continuous pain, the tenderness along the entire nerve, and the presence of paresthesia, anesthesia, paresis, and wasting will serve to distinguish neuritis from neuralgia.

The *lightning-pains of locomotor ataxia* must not be mistaken for neuralgia. The ataxia, Argyll-Robertson pupil, girdle sensation, bladder disturbances, and absence of knee-jerk will serve to exclude neuralgia.

Prognosis.—For the attack the prognosis is good; for permanent cure, it must be guarded. If the cause can be removed, the prognosis is favorable.

Treatment.—*The Interval.*—Careful search should be made for an exciting cause, which, if found, must be removed. The teeth, eyes, nose, gastro-intestinal tract, urine, and blood should be carefully examined.

If the disease is associated with anemia, iron and arsenic are indicated. If there is any suspicion of syphilis, mercury and iodids should be tried. If a malarial element is present, quinin may effect a cure. In gouty subjects much may be expected from regulation of diet, systematic exercise, and the administration of alkalis. In chronic lead-poisoning iodids are serviceable.

All influences that tend to induce a morbid excitability of the nervous system—mental or physical fatigue, emotional excitement, sexual excesses, overindulgence in tobacco, tea, coffee, or alcohol—should be avoided.

In every case an endeavor must be made to improve the general nutrition, which is almost always disturbed. The measures to be employed for this purpose include an abundance of fresh air, proper food, regular hours, adequate protection from the vicissitudes of weather, systematic exercise, frequent bathing with friction, and the use of such tonics as iron, arsenic, and codliver oil.

The following combination is sometimes useful:

℞. Quininae sulphatis.....	gr. xxiv
Arseni trioxidi.....	gr. ss
Ferri reducti.....	℥ss
Calcii glycerophosphatis.....	℥iss.—M.
Pone in capsulas No. xxiv.	
SIG.—One capsule after meals.	

In refractory trigeminal neuralgia recourse may be had to the injection of alcohol (80 to 90 per cent.) into the main trunks of the nerves. This measure usually affords relief for a period of from eight months to a year. Finally, after failure with all other measures, removal of the sensory root of the Gasserian ganglion should be considered. This operation almost always affords permanent relief and in skillful hands the risk is small (less than 1 per cent.).

The Attack.—Heat, dry or moist, may be applied for its soothing effect. A liniment of aconite or of chloroform is sometimes efficacious. Menthol and chloral-camphor are useful in neuralgia of superficial nerves if the pain is slight. Acupuncture and aquapuncture are effective, but are not suitable for use about the face. In obstinate cases active counterirritation by means of blisters or the thermocautery will sometimes be found a potent remedy. In trifacial neuralgia the blisters may be applied behind the ear.

Among the internal remedies most worthy of confidence may be mentioned acetphenetidin or antipyrin (5 to 10 grains), bromids, cannabis indica, croton chloral, caffein, gelsemium, and salicylic compounds. Morphin is undoubtedly the most certain means we possess of affording temporary relief, but on account of the danger of inducing the opium habit it should be employed only as a last resort. Combinations of a bromid with acetphenetiden may often be prescribed advantageously. Croton chloral, in doses of from 5 to 10 grains, and tincture of gelsemium, in doses of 10 minims or more, are occasionally serviceable in trifacial neuralgia. Caffein (2 to 3 grains) is sometimes efficacious. Combinations of caffein with acetphenetidin or with bromids in many cases do more good than single drugs. Neuralgia brought on by exposure to cold and wet is favorably influenced by the salicylates. In such cases, the following combination will be found of value:

R. Acetphenetidini
 Salophen..... aa ʒiss
 Codeinæ sulphatis..... gr. iij.—M.
 Fiant chartulæ No. xii.
 SIG.—One powder every two or three hours.

DISEASES OF THE MUSCLES AND JOINTS

MYALGIA

Myalgia is an affection of the voluntary muscles characterized by pain, which is aggravated by movement. The pain varies in degree from a dull ache to an intense cramp. Malposition and even contractures may result from the efforts of the patient to avoid moving the affected muscles. The nature of the disease is obscure. It is probably in some cases a form of myositis or of neuritis affecting the nerve-endings in the muscles. Various names have been given to it according to the different groups of muscles involved. The most frequent forms are: *Wry-neck* (torticollis) in which the head is fixed and rotated toward the unaffected side; *pleurodynia*, in which severe pain is experienced in the intercostal muscles upon coughing or turning the body; and *lumbago*, in which the lumbar muscles are painful and immobile. The condition may be acute or chronic.

Acute myalgia is usually due to overexertion or to chilling and wetting, especially when the body is overheated. Many writers lay stress upon a "rheumatic" or gouty diathesis. It is sometimes associated with acute articular rheumatism.

Chronic myalgia may be caused by (1) various intoxications (alcohol, lead, diabetes, gout, etc.); by (2) chronic infections (focal septic infection, syphilis, tuberculosis); by (3) unusual strain from a lack of muscular balance; or (4) lesions of bones, joints and viscera, the nerves of which are in relation with those of the affected muscles.

Chronic lumbago may be a result of (1) disease of the spine or sacroiliac joints; (2) defective muscular balance (faulty

posture, spinal curvature, flat foot, shortening of one leg, etc.); (3) traumatism; (4) abnormal abdominal or pelvic conditions (visceroptosis, renal calculus, retroversion or prolapse of uterus, ovarian disease, prostatic lesions, etc.); (5) chronic focal infection in the tonsils, teeth or elsewhere; (6) chronic intoxications (lead, diabetes, gout, ect.); and (7) neurasthenia or hysteria.

Treatment.—In acute cases anodynes are required. A combination of acetylsalicylic acid and acetphenetidin, with or without codein, usually suffices.

℞. Acidi acetylsalicylici
 Acetphenetidini..... āā ʒj
 Codeinæ sulphatis..... gr. ij-ijj.—M.
 Fiat chartulæ No. xii.
 SIG.—One powder every 3 or 4 hours.

Locally, heat, support by adhesive straps, dry cupping, or friction with a stimulating liniment will afford much relief.

℞. Methylis salicylatis..... fʒj
 Linimenti chloroformi..... fʒiv.—M.
 S.G.—To be rubbed in over the painful part.

Persistent myalgia is sometimes favorably affected by massage and high-frequency electric currents.

In chronic myalgia treatment must be directed to the underlying cause. Cases of lumbago due to sprain, arthritis or sacro-iliac relaxation will require immobilization of the parts by straps of adhesive plaster or specially constructed corsets. Unilateral defects in balance are sometimes corrected by elevating one heel. In septic cases the primary source of the infection must be removed. Backache resulting from relaxed muscles and postural curve will be benefited by the assumption of a correct standing and sitting position, gymnastic exercises, massage, shoulder-straps, etc. In severe cases, irrespective of the cause, recumbency for a part of the day is often necessary at first.

MUSCULAR DYSTROPHIES

Definition.—An atrophic condition of the muscles developing in early life and not dependent upon any lesion in the nervous system.

Etiology.—The disease usually manifests itself before puberty. It is more common in males than in females. It is frequently transmitted from generation to generation, and several members of the same family may be similarly affected.

Pathology.—No lesion in the cord or nerves is observed. Gowers regards the disease as of developmental origin. Microscopic examination of the muscles reveals atrophy of their fibers and an unnatural amount of fat and connective tissue. When the latter elements are considerably increased, a pseudohypertrophy results (pseudomuscular hypertrophy).

Varieties.—The following types are recognized: (1) Pseudomuscular hypertrophy; (2) Erb's juvenile dystrophy; (3) Landouzy-Dejerine type.

Pseudomuscular Hypertrophy.—This form begins in childhood between the second and seventh years. The first symptom to attract attention is weakness of the muscles; the child is awkward, stumbles, and in walking seeks support. As the paralysis increases the muscles, particularly those of the calf, thigh, buttock, and back, enlarge. The upper extremities are less frequently affected. When the child assumes the erect posture, the feet are wide apart, the belly protrudes, and the spinal column shows a marked curvature with the convexity forward. The manner of rising from the recumbent position is characteristic: The patient straightens himself either by grasping the knees or by resting the hands on the floor in front of him, extending the legs, and pushing the body backward. The gait is waddling in character.

The electric contractility of the muscles is gradually reduced, but the reaction of degeneration is not present. There are no fibrillary twitchings in the muscles. The knee-jerk

is lessened or abolished. There are no mental or sensory disturbances.

In the course of years the atrophy becomes so marked that the patient is unable to leave his bed; the enlargement of the muscles is followed by atrophy; and finally death results from some intercurrent disease or inflammation of the lungs induced by the weakened respiratory power.

Erb's Juvenile Dystrophy.—This form usually develops between the ages of twelve and sixteen. The muscles of the shoulder are first affected. The wasting may be preceded by hypertrophy.

Landouzy-Dejerine Type.—This type usually develops in early childhood and is characterized by bilateral atrophy of the facial muscles. It differs from *bulbar palsy* in that it does not involve the tongue or the muscles of deglutition.

Diagnosis.—In *chronic poliomyelitis* (progressive muscular atrophy of spinal origin) the symptoms come on later in life, hereditary or family influences are rarely present, the small muscles of the hand are first affected, and the wasting is associated with fibrillary twitchings. In *multiple neuritis* paralysis precedes the wasting, sensory symptoms are usually present, and the history reveals a definite cause.

Prognosis and Treatment.—The disease is incurable, but the progress is slow. Massage, electricity, and graduated exercise may be followed by temporary improvement.

CONGENITAL MYOTONIA

(Thomsen's Disease)

This is a disease confined to certain families, and characterized by tonic spasm of the muscles upon every attempt at voluntary movement after a period of rest. Persistence in movement results in relaxation of the muscles, which continues while they are in use. The muscles are well developed, and are unduly irritable, quick blows and mild electric currents producing contractions that are tetanic in character. With the galvanic current the anode-closing contraction becomes equal to or greater than the cathode-closing contraction. The disease develops in childhood and continues through life.

CONGENITAL AMYOTONIA

(Congenital Myatonia ; Oppenheim's Disease)

This is a congenital disease characterized by flaccidity and loss of tone and power in the voluntary muscles, without absolute paralysis or atrophy of the muscles. The deep reflexes are diminished or lost and the electric excitability of the muscles is impaired. The condition usually improves as the child grows older but complete recovery is rare. It differs from the muscular dystrophies in being congenital and non-familial, and in the absence of wasting.

MYASTHENIA GRAVIS

(Asthenic Bulbar Palsy)

In this disease the muscles are rapidly exhausted upon slight exertion, but recover after a brief rest. All the muscles of the body may be affected, but those supplied by the cranial nerves are most frequently involved, the symptoms simulating those of true bulbar palsy (inferior polioencephalitis). There is no atrophy or reaction of degeneration, but with the faradic current, there is early exhaustion of the muscles (myasthenic reaction). The disease usually lasts for several years, and remissions are common. Death may occur from disturbances of respiration or deglutition. The treatment consists in rest, careful feeding (nasal feeding if necessary) and the use of strychnin and other tonics. Massage and electricity are contraindicated.

ARTHRITIS DEFORMANS

(Rheumatoid Arthritis; Osteo-arthritis)

Definition.—A form of arthritis, chronic throughout or with an acute onset or acute exacerbations, leading to more or less permanent deformity and crippling of the joints, and probably dependent upon a local focus of infection. The disease is in no way related to either rheumatism or gout.

Etiology.—Arthritis deformans may occur at any age, but it is uncommon in the extremes of life. Females are attacked more frequently than males. An hereditary tendency to the disease is sometimes noted. Enfeeblement of the general health from mental strain, overwork, unsanitary surroundings, frequent pregnancies, etc., is an important etiologic factor.

In many cases the disease can be definitely associated with some local infection, such as tonsillitis, otitis media, pyorrhea alveolaris, dental abscesses, cystitis, gonorrhea, intrapelvic suppuration, or an infected wound. Occasionally trauma seems to stand in a causal relationship.

Pathology.—In one form (periarticular) the changes affect chiefly the soft tissues, and consist of serous effusion into the joint, thickening of the synovial membrane and surrounding structures, and slight erosion of the cartilages. In another form (atrophic) the cartilages show extensive destruction, the heads of the bones are bare and eburnated, and the shafts are rarefied. In a third form (hypertrophic) there is much new bone-formation (osteophytes) at the edges of the cartilages, and sometimes also in the surrounding fibrous tissues, causing great deformity. In many cases the changes are mixed and do not correspond exactly to any one form.

Secondary lesions are not uncommon. These include atrophy of the muscles, contractures, partial luxations, and localized neuritis.

Symptoms.—When the onset is acute the disease resembles rheumatism, but in contrast to the latter the smaller joints, especially those of the hands, neck, and jaw, are often involved, the inflammation rarely shifts from joint to joint, the temperature seldom exceeds 102° F., the pulse-rate is usually elevated out of proportion to the fever, endocarditis is exceptional, the salicylates are of little value, and, above all, there is a marked tendency to structural changes in the joints and muscular atrophy.

In the form with a chronic onset one or two joints are, as a rule, first attacked, and then the disease gradually becomes more or less general. Pain, swelling, and impaired mobility are, for a time, the chief symptoms, but sooner or later signs of structural changes show themselves. These comprise deformity, rigidity, and crepitation on movement. Muscular atrophy is also a constant feature, and very often contractures or partial luxations add to the deformity. In

advanced cases the joints are usually fixed in a position of flexion, though in the hands the terminal row of phalanges are frequently extended. Acute exacerbations are very common. Certain general features, such as anemia, an increased pulse-rate, profuse sweating about the hands and feet, and irregular pigmentation of the skin are sometimes observed.

Monarticular Form.—This form occurs chiefly in old persons, and usually affects either the hip or shoulder. The symptoms are persistent pain, impaired mobility, and muscular atrophy.

Spondylitis Deformans.—This term is applied to arthritis deformans of the spine; other joints may or may not be involved. The chief symptoms are pain in the back or in the limbs, especially the legs; limitation of motion, and ultimately extreme stiffness or fixation of the spine ("poker-spine"), exaggerated reflexes, gradual muscular wasting, and, in some cases, changes in the spinal curve or undue prominence of the spine. The *x*-ray picture is a valuable aid to diagnosis. The disease is a common cause of sciatica and lumbago.

Heberden's Nodes.—These are small nodules at the sides of the terminal phalanges of the fingers; they are not often painful; they are sometimes the sole expression of mild arthritis deformans, but they apparently occur also in gout.

Diagnosis.—The differential diagnosis between rheumatoid arthritis and *gout* has been considered (see p. 424).

Prognosis.—Rheumatoid arthritis, though usually a progressive disease, sometimes remains limited to one or two joints or becomes arrested. A complete cure is exceptional.

Treatment.—In acute attacks the treatment is that advised for acute rheumatism. In chronic cases the removal of foci of infection, if such can be found, and attention to improving the general nutrition of the patient by good food, pure air, rest, bathing, freedom from worry, etc., stand first in importance. A change of residence to a dry, warm, and equable climate is often desirable. Hot sulphur and hot saline baths are frequently useful. Tonics, such as iron, arsenic, and cod-

liver oil are sometimes of service. Potassium iodid is occasionally beneficial. Vaccines, especially autogenous, and even non-specific proteins (10 million killed typhoid bacilli, increased to 1 or 2 billion every 4 to 8 days) are often of value. Thyroid extract (1 to 2 grains twice daily) may be tried. Salicylates and cinchophen sometimes relieve pain, but have no influence on the disease.

Local Treatment.—Massage and graduated exercise, often accomplish much good, but should not be used during acute phases. Super-heated air-baths are sometimes very useful. Rubefacient liniments have a palliative influence in mild cases. An ointment of mercury, belladonna, and ichthyol, or an ointment of iodine well rubbed into the affected part is sometimes efficacious. If the pain is severe and persistent, blisters, or light applications of the actual cautery may prove effective.

CHRONIC RHEUMATISM

Cases of rheumatic fever are not infrequently met with which drag on for weeks or months. To these the term *subacute rheumatism* is applicable. There is no proof, however, that rheumatism ever passes into a distinctly chronic condition or ever begins as a chronic affection. The many cases of polyarthritis with a tendency to chronicity and to permanent structural changes in or about the joints, which are usually referred to as chronic articular rheumatism, had better be regarded as examples of *rheumatoid arthritis*, and treated accordingly, until we possess a more accurate knowledge of their etiology and can classify them more definitely.

VASOMOTOR AND TROPHIC DIS- ORDERS, SUNSTROKE, AND INTOXICATIONS

RAYNAUD'S DISEASE

(Symmetric Gangrene)

Definition.—A vasomotor neurosis characterized by attacks of local anemia, congestion, and gangrene in the projecting parts of the body.

Etiology.—The cause is unknown. The disease is believed to be dependent upon spasm of the peripheral arterioles of central origin. Exposure to cold, even of moderate degree, is a common exciting factor.

Symptoms.—In the first stage the affected part becomes extremely pale, cold, and anesthetic (*local syncope*). After a variable time the part becomes purple, livid, and intensely painful (*local asphyxia*). Such attacks may be excited by cold, and come and go without damaging the part. Sometimes the disease advances to the third stage, in which congestion gives way to *dry gangrene*. Symmetric parts, as a finger on each hand, a toe on each foot, or the lobes of the ears, are usually affected. Hemoglobinuria may occur in, or replace, an attack.

Prognosis.—The attacks persist, but life is rarely endangered.

Treatment.—Patients liable to attacks should be well protected against cold. Tonics are often indicated. Frequent bathing followed by friction is useful. Raynaud advises the use of the continuous current—one pole over the spine and the other over the affected area. Good results

have been reported in some cases from the use of high frequency currents. Nitroglycerin may prove useful.

ANGIONEUROTIC EDEMA

This affection, which is usually regarded as a vasomotor neurosis, but which in reality is often an anaphylactic phenomenon, is characterized by the acute development of circumscribed swellings in the subcutaneous tissues and mucous membranes in various parts of the body, lasting from a few hours to several days, and showing a marked tendency to recur. The face is the part most frequently affected. Coincident with the edema there may be disturbances of the gastrointestinal tract such as nausea, vomiting, colicky pain, and diarrhea. In some instances the swelling is accompanied by burning and itching, and an outbreak of urticaria. Purpura may also occur.

The disease is most frequently observed in neuropathic subjects and is sometimes hereditary. The attacks may be excited by exposure to cold or the ingestion of certain articles of food (shell-fish, strawberries, etc.) poisonous to the individual. In a few instances death has resulted from edema of the larynx. Removal of the cause, if possible, regulation of the diet, and the judicious use of saline laxatives are important elements in treatment. Among special remedies that have been recommended may be mentioned calcium chlorid (10 to 15 grains thrice daily), nitroglycerin, strychnin, quinin, and arsenic.

HEAT STROKE

(Sunstroke; Thermic Fever; Insolation; Heat Exhaustion)

One of two conditions may ensue as a result of exposure to excessive heat—sunstroke or heat exhaustion. Lowered vitality from other diseases, bodily fatigue, and alcoholic excess are important predisposing factors. It is probable that the excessive heat leads to the formation of certain toxic

substances that disturb the heat-regulating centers or the vasomotor centers in the brain.

Sunstroke (Thermic Fever).—This condition occurs after exposure to the sun's rays. Premonitory symptoms consisting of headache, dizziness, nausea, and malaise are sometimes present, but not rarely there is sudden coma with high fever (105 to 110° F. or more), flushed face, dry skin, rapid pulse, and stertorous breathing. Muscular twitchings and epileptiform convulsions are not uncommon. Death may occur within a few hours or the temperature may fall, the coma disappear and recovery gradually ensue. Chronic meningitis, persistent headache, impairment of memory, and extreme sensitiveness to high temperature are frequent sequelæ. The mortality is probably not less than 40 per cent. In fatal cases the pathologic changes consist of congestion of the various organs, especially the brain, abnormal fluidity of the blood, and parenchymatous degeneration of the heart, liver, and kidneys. The conditions under which the coma has occurred, with the intense fever will usually serve to distinguish sunstroke from *apoplexy*, *alcoholism* and *uremia*.

Treatment.—The patient should be placed in a bath of ice-water and rubbed with ice. Ice-water enemas may also be employed. The intravenous or subcutaneous injection of salt solution has proved beneficial in some cases. Packard and others have found venesection (10 to 20 ounces) very effective in asphyxiated subjects. Weakness of the pulse is not necessarily a contraindication, as the circulation often improves during the operation.

Heat Exhaustion.—This condition results from exposure to either natural or artificial heat. It is characterized by profound prostration, a subnormal temperature, a feeble pulse and collapse. Complete syncope may occur or there may be semiconsciousness with muttering delirium. The prognosis is usually favorable. The treatment consists in the use of external heat and the administration of stimulants, such as camphor, strychnin, ammonia, and whiskey.

ALCOHOLISM

(Inebriety)

Acute Alcoholism.—The ingestion of a large quantity of alcohol produces the following symptoms: Flushing of the face, mental excitement, quickening of the pulse and respiration; then incoherent speech, delirium, dilated pupils, loss of coördination, subnormal temperature, vomiting, and, finally, stupor and coma. Not infrequently the coma is interrupted by convulsive seizures. In most cases, if the dose has not been too large, recovery follows in a day or two.

Chronic alcoholism is characterized by disturbed sleep, fine tremors, mental impairment, injection of the eyes, redness of the nose, and the symptoms of gastro-intestinal catarrh. When the habit is long continued, degenerative and cirrhotic changes in the heart, blood-vessels, liver, and kidneys are likely to develop.

A common complication of chronic alcoholism is *delirium tremens*, which is usually excited by temporary excess, abstinence from food, an injury, or some acute intercurrent disease, especially pneumonia. It is manifested by great mental excitement, apprehensiveness, insomnia, incoherent speech, tremors, disordered intellect, and terrifying hallucinations of sight or hearing. The pulse is rapid and feeble, the appetite is lost, the bowels are constipated, and the temperature is slightly elevated. In favorable cases convalescence follows in a few days, but not rarely stupor succeeds the delirium and symptoms of the so-called typhoid state supervene. This condition, which depends upon an excessive accumulation of serous fluid in the pia-arachnoid space ("wet brain"), may persist for several weeks.

Among other sequels of dipsomania may be mentioned pneumonia, chronic meningitis, cirrhosis of the liver, arteriosclerosis, multiple neuritis, amblyopia, epilepsy, and dementia.

Diagnosis.—The *coma of alcoholism* must be distinguished from the coma of other diseases. The history, the absence

of paralysis, the subnormal temperature, the fact that the patient can usually be aroused, the odor on the breath, and the absence of other cause will usually prevent an error in diagnosis.

Delirium tremens is recognized by the history, restlessness, delirium, tremors, and terrifying hallucinations.

Prognosis.—*In acute alcoholism* the prognosis is guardedly favorable. In *delirium tremens* after the occurrence of actual hallucinations the mortality is not less than 20 per cent., and after the development of cerebral edema not less than 75 per cent. In *chronic alcoholism* the outlook is not very favorable. Temporary improvement is only too often followed by relapse.

Treatment.—*Acute Alcoholism.*—The stomach should be emptied by the stomach-pump, a stimulating emetic, or the hypodermic injection of apomorphin ($\frac{1}{10}$ grain). If the coma persists and the pulse weakens, cardiac stimulants, such as ammonia, strychnin, and digitalis, should be administered hypodermically. Douching and flagellation may also be employed to arouse the patient.

Treatment of Delirium Tremens.—As there has usually been a complete abstinence from food during the debauch leading to the delirium, easily digestible foods are always necessary, and the best are milk with lime-water, eggs, and highly seasoned beef-tea. Sleep must be secured by chloral (20 grains), scopolamin ($\frac{1}{100}$ grain), potassium bromid (1 dram), or paraldehyd (1 fluidram). Active catharsis should be encouraged. If the pulse is weak, strychnin and digitalis will be found useful stimulants. In many cases physical restraint will be required; it may be secured by strapping the patient to the bed with sheets. Should profound stupor develop, the application of a blister to the back of the neck, a few light touches of the actual cautery, or lumbar puncture will often serve to arouse the patient.

Chronic Alcoholism.—It is necessary that alcohol shall be withdrawn; the rapidity with which this can be accomplished will depend on the circumstances. In most cases the tempta-

tion to drink is so strong that confinement in an inebriate asylum is essential to the success of the treatment. Various substitutes have been recommended for alcohol, among which may be mentioned bromid of potassium, chloral, scopolamin, and cannabis indica. These accomplish little beyond quieting the patient and securing sleep. The diet should be nutritious, and carefully adapted to the condition of the stomach, which is usually the seat of chronic catarrh. Tonics are often indicated. Graduated physical exercise is sometimes of decided value.

CHRONIC OPIUM POISONING

(Morphinomania)

Symptoms.—The symptoms resulting from the habitual use of opium are an irresistible craving for the drug, loss of flesh and strength, tremors, anemia, a peculiar sallow complexion, anorexia, deranged digestion, a tendency to diarrhea, disturbed sleep, mental depression, irritability, loss of will power, and moral perversion.

Treatment.—Isolation in a special institution or asylum is almost imperative. As a rule, the drug should be withdrawn rapidly, but in aggravated cases not too abruptly, for fear of collapse. The diet should consist of nutritious, easily digested food. Strychnin, while it is without specific action, is often valuable for its stimulating effect. Bromids, acetphenetidin, salicylates, and cannabis indica are sometimes useful in ameliorating the distress that follows the withdrawal of opium. Trional, barbital, paraldehyd, and scopolamin are the best hypnotics. Massage, graduated exercise, and the Turkish bath are useful roborant measures in the convalescent stage.

CHRONIC LEAD-POISONING

(Plumbism; Saturnism)

Etiology.—Chronic lead-poisoning may be brought about by the too prolonged use of the salts of lead for medicinal

purposes, but it is much more frequently induced in workmen who are exposed to the fumes or dust of lead, or who handle the metal or paints containing it. It may follow, also, the accidental introduction of lead into the system through drinking-water, articles of food, hair-dyes, and cosmetics. Occasionally it results from the use of water that has been carried through lead pipes or has been stored in cisterns lined with lead.

Symptoms.—The following are the chief manifestations: Anemia with granular degeneration (basophilic) of the red cells; severe colicky pains centering around the umbilicus, with retraction and rigidity of the abdominal wall; constipation; a blue line on the gums near the insertion of the teeth, due to the deposition of a sulphuret of lead; paralysis; and occasionally grave cerebral symptoms (encephalopathies), tremors, and pains in the joints (arthralgia.) Important sequels are gout, arteriosclerosis and chronic nephritis.

The Paralysis.—This usually attacks the muscles supplied by the musculospiral nerve—the extensors of the fingers and of the wrist—causing the so-called “wrist-drop.” The affected muscles ultimately atrophy and yield the reactions of degeneration. Occasionally other muscles are involved, such as the extensors of the legs, the recti of the eye, and the adductors of the larynx. Sensation is not affected.

Encephalopathies.—These are among the more rare manifestations of plumbism, and consist of convulsions, coma, delirium, intense headache, and blindness from atrophy of the optic nerves.

Prognosis.—The outlook is usually favorable except in cases with encephalopathies.

Treatment.—Prophylaxis consists in absolute cleanliness; the use of respirators in lead factories; the avoidance of eating in an atmosphere laden with the dust of the metal; and in the occasional use of Epsom salts.

The curative treatment consists in the administration of iodid of potassium (10 grains thrice daily) and the use of

sulphur baths. Constipation should be relieved by Epsom salts. The colic may require the hypodermic injection of morphin and atropin and the application of hot fomentations to the abdomen. For the paralysis massage, electricity and strychnin should be used.

BOTULISM

Botulism is a form of poisoning caused by a toxin which is produced in foods outside of the body by the spore-bearing anaërobe, *Clostridium botulinum*, which is widely distributed in nature. Boiling for five minutes destroys the toxin, but the spores are very resistant. The majority of outbreaks of botulism in this country have been caused by canned vegetables or fruits, but sausages and potted meats have frequently been the source of the poisoning.

The *symptoms*, which usually develop in from 12 to 72 hours after the contaminated food has been eaten and which are apparently due to thrombotic occlusion of small vessels and a secondary inflammatory reaction in the brain-stem, consist of vertigo, ocular palsies (ptosis, diplopia, etc.), difficulty in deglutition and phonation, and extreme muscular weakness. Vomiting sometimes occurs at the onset. Fever is absent. The average mortality in 150 cases was about 65 per cent. Death, which usually occurs within a few days, is caused by respiratory or circulatory failure, or by aspiration pneumonia, as in bulbar palsy.

Treatment.—Gastric lavage, free catharsis, circulatory and respiratory stimulants, oxygen inhalations, and feeding, if necessary, by the stomach tube are important therapeutic measures. An antibotulinus serum may be helpful.

DISEASES OF THE SKIN AND ITS APPENDAGES

THE COLOR OF THE SKIN

Pallor as a permanent condition is usually an expression of anemia; but it should be borne in mind that the surface may be pale when the blood is normal and, on the other hand, that a normal color of the skin may coexist with marked anemia.

Yellowness of the skin may result from *jaundice*, in which case the conjunctivæ will also be yellow and the urine will contain bile. Yellowness may also result from *chlorosis*, *pernicious anemia*, and the *cachexia of cancer*.

Whiteness of the Skin.—A milk-white hue over extensive areas may be observed in *albinism*, *vitiligo*, and in *leprosy*.

Dark-brown or gray discoloration of the skin may be observed in the following conditions: *Addison's disease*, *hemachromatosis* (bronzed diabetes), *chronic poisoning with silver nitrate* (*argyria*), *melanosarcoma of the skin*, *chronic malaria*, *chronic jaundice*, *pernicious anemia*, *splenic anemia*, *Graves' disease*, *pellagra*, and so-called *vagabond's disease*, the last being a discoloration resulting from exposure, uncleanliness, and irritation caused by pediculi.

Blueness of the skin as a permanent condition is usually an expression of cyanosis.

HARDNESS OR INDURATION OF THE SKIN

Induration of the skin is observed in *scleroderma*. In this affection the skin is tense, hide-bound, and more or less pigmented. Induration is also observed in *myxedema*. In this condition the skin is swollen, as in edema, but it is firm, in-

elastic, and does not pit on pressure. In addition the features are peculiarly broadened and the mental power is impaired. *Edema*, or *dropsy of the subcutaneous tissues*, if extreme and persistent, may also cause induration.

GLOSSY SKIN

"Glossy Skin."—This term was applied by Paget to indicate a smooth, atrophied, and shiny appearance of the skin. It is most frequently observed after inflammation or injury of the nerve-trunks. It is sometimes associated with an intense burning pain to which Mitchell has given the name *causalgia*.

ENLARGEMENT OF THE SUPERFICIAL VEINS

Enlargement of the superficial veins may result from chronic heart, lung, or liver disease or from the pressure of a tumor or aneurysm on deep-seated veins. As a general condition it may be congenital and result from occlusion of the deep-seated veins.

CUTANEOUS EMPHYSEMA

This term is applied to the presence of air or gas in the subcutaneous tissues. The condition is manifested by a diffuse swelling which yields a peculiar crackling sensation on palpation. It may result—(1) From the rupture through mechanical injury or ulceration of some air-containing structure, such as the trachea, lung, esophagus, stomach, or bowel; or (2), in rare instances, from the presence in the tissues of gas-producing bacteria, as in malignant edema.

ABNORMAL CONDITIONS OF THE NAILS

Atrophy of the Nails.—The nails may become dry, brittle, discolored, and cracked in organic disease of the spinal cord; after inflammation or injury of the peripheral nerves; after prolonged febrile diseases, such as typhoid fever; and in certain affections of the skin that involve the matrix of the nail, such as eczema, psoriasis, and ringworm.

Curving of the Nails.—Incurvation of the nails is usually associated with clubbing of the terminal phalanges. It may be observed in pulmonary tuberculosis, bronchiectasis, chronic empyema, chronic heart disease and in many wasting diseases.

Onychia.—Inflammation of the matrix of the nail may result from injury; from syphilis; from organic disease of the spinal cord, as locomotor ataxia; and from cutaneous affections involving the matrix, as leprosy, ringworm, and eczema.

CUTANEOUS ERUPTIONS

Macules.—Macules are discolored spots that are neither elevated nor depressed.

A general red macular eruption is observed in the following conditions:

Syphilis.—Secondary syphilis may manifest itself as an eruption of small red macules. They are usually abundant and frequently cover the entire body; they are unattended by subjective symptoms; they are usually associated with the history or with the evidences of syphilis, such as the scar of the chancre, bone-pains, alopecia, swollen glands, and sore throat.

Erythema multiforme may manifest itself as a macular eruption, but the macules are usually associated with dark-red papules or tubercles. The multiformity of the lesions; their preference for the extremities; their symmetrical distribution; the short duration of each lesion; the absence of marked itching; and the presence of rheumatoid pains are the diagnostic features.

Pityriasis Rosea.—The eruption is especially found on the trunk; the lesions are of a rosy-red or salmon tint; they are slightly scaly, the scales being dry; itching is slight or absent; and the duration is a few weeks.

Rubella.—This affection produces a macular or maculopapular rash that disappears in two or three days with slight desquamation. The moderate fever, sore throat, swollen

cervical glands, and history of contagion will assist in the diagnosis.

Toxic Rashes.—The ingestion of certain drugs (antipyrin, arsenic, quinin, therapeutic serums) and, in some persons, of certain articles of food ("shell-fish," strawberries) is followed by the appearance of a red macular eruption.

Brown macules are observed in:

Lentigo, or Freckle.—The spots are small, and are found especially on exposed parts—face, neck, shoulders, and hands.

Chloasma.—Dark spots may result from irritation of the skin from the action of chemicals, heat, scratches, or blisters. They are sometimes noted in general diseases, such as Addison's disease and syphilis. They also occur in primary affections of the skin, as vitiligo, scleroderma, and leprosy.

Mole, or Nævus Pigmentosa.—Moles consist in congenital deposits of pigment on various parts of the body.

White macules are observed in:

Vitiligo.—Apart from the absence of pigment, the skin is normal in appearance and function. An excess of pigment is usually noted at the periphery of the white patches.

Leprosy.—In this condition there are structural changes in the skin and anesthesia in addition to the white appearance.

Morphea.—In the late stage of this affection the circumscribed patches are white or yellow. The structure of the skin is altered, and the periphery of the patches is distinctly hyperemic.

Purpuric spots, petechiæ, or ecchymoses occur in many diseases and intoxications (see p. 176) and may also result from the bites of various insects (fleas, bedbugs, body-lice). In purpuric spots of parasitic origin the central bite-mark, the surrounding hyperemic zone and the presence of the insects are the diagnostic features.

Diffuse erythema may result from:

The Action of Certain Drugs (Dermatitis Medicamentosa). Belladonna, quinin, chloral, cubebs, salicylic acid sulphonal, and arsenic may produce a diffuse red rash.

Scarlet Fever.—The history of contagion, high fever, sore throat, swollen glands, rapid pulse, and the punctiform character of the rash will indicate the diagnosis.

Rubella.—In some cases of rubella the eruption is red and diffuse. The history, slight fever, mild catarrh, and marked swelling of the postcervical glands will suggest rubella.

Local irritation from traumatism, excessive heat, x-rays, poisonous plants, or drugs often produces erythema.

Erythema Intertrigo.—This occurs where two cutaneous surfaces come in contact. The part is red, moist, and sometimes macerated. The condition excites a burning pain.

Eczema.—The skin is thickened and infiltrated; there is marked itching; the redness shades off gradually; and there is no fever.

Erysipelas.—The part is considerably swollen; the redness and swelling terminate in an abrupt ridge and the temperature is high.

Acne Rosacea.—This is a chronic disease; the redness appears on the face and is associated with acne lesions and dilated capillaries.

Pellagra.—The erythematous patches develop in the spring and subside in the winter; they occur especially on the hands, feet, and neck; and they are associated with digestive disturbances, spasmodic tabes, and often with insanity.

Vesicles.—Vesicles are minute epidermic elevations containing serous fluid. They are observed in the following conditions:

Sudamen.—This consists of an eruption of minute vesicles that result from the imprisonment of sweat in the layers of the skin. It is usually associated with free perspiration; the vesicles are translucent, lack inflammatory characteristics, and show no tendency to rupture.

Herpes.—The vesicles appear in groups or clusters; they are mounted on an inflammatory base; they show no tendency to rupture; they are frequently associated with burning

or neuralgic pains; and they are distributed along the line of the nerve-trunks.

Dermatitis Venenata.—A vesicular eruption may result from contact with poisonous plants, such as the poison-ivy or poison-oak. The eruption usually appears on the exposed parts—face or hands; the part is red and swollen and there is intense itching.

Dermatitis Herpetiformis.—The vesicles are often irregular in shape; they appear in clusters; they are very tense; they show no tendency to rupture; they are frequently associated with other lesions—papules, pustules, and bullæ; they excite intense itching; and they appear in crops over a period of weeks or months.

Impetigo Contagiosa.—The eruption consists of small vesicles that subsequently enlarge until they reach the size of blebs; the vesicles appear in crops; are commonly discrete; are flat and umbilicated; are filled with a straw-colored fluid; they show no tendency to break, but dry up and form thin yellow crusts, and they excite but little itching. The disease is contagious and auto-inoculable; occurs especially in children; and lasts from ten days to two weeks.

Vesicular Eczema.—The vesicles are quite small and are aggregated in patches; the intervening skin is red and thickened; the vesicles tend to break and pour forth a serous fluid that keeps the part moist; and the eruption is associated with intense itching.

Miliaria, or Heat-rash.—This may appear as an eruption of minute vesicles; they are always discrete; they are surrounded by red areolæ; they usually appear on the trunk; they are generally associated with pin-head papules; they show no tendency to rupture; and they excite a little burning and itching.

Scabies.—In this affection the vesicles are small; they are usually associated with pustules and *burrows*; they excite intense itching; and they are usually found on the hands, forearms, in the axillæ, under the mammæ, and on the inner aspects of the thighs.

Blebs, or Bullæ.—A bleb, or bulla, is a circumscribed elevation of the skin containing serous fluid, and varying in size from a pea to an egg. Blebs are observed in the following conditions:

Impetigo Contagiosa.—The blebs are flat and umbilicated; they contain a straw-colored fluid; they appear in crops; they are commonly discrete; they show no tendency to break, but dry up and form thin yellow crusts; and they excite but little itching. The disease is contagious and auto-inoculable, occurs especially in children, and lasts from ten days to two weeks.

Dermatitis Herpetiformis.—The bullæ are frequently associated with papules, vesicles, and pustules; they are surrounded by inflamed skin; they appear in clusters; they show no tendency to break, but dry up and leave yellowish-brown crusts; and they excite considerable itching.

Pemphigus.—The bullæ appear in crops; excite but little itching; they lack an inflammatory areola; and, as a rule, they dry up, and leave behind a thin pellicle. The disease is generally chronic.

Syphilis.—The bullous syphilid is observed in hereditary syphilis and very late in the acquired disease. The contents of the bullæ soon become pustular; the blebs dry up, and form dark-green, cone-shaped, stratified crusts, which become detached and leave discharging ulcers. The history and the other evidences of syphilis will aid in the diagnosis.

Pustules.—A pustule is a small circumscribed elevation of the skin containing pus. Pustules are observed in the following diseases:

Eczema Pustulosum.—The pustules are small; are aggregated in a patch; are generally associated with minute vesicles; the intervening skin is red and thickened; and there are marked burning and itching.

Acne Vulgaris.—The pustules are usually confined to the face, back, and shoulders; they have their origin in the seba-

ceous follicles; they are usually associated with papules and comedones; and they excite no itching.

Dermatitis Herpetiformis.—The pustules are frequently associated with papules and vesicles; they are surrounded by inflamed skin; they appear in clusters; and they excite considerable itching.

Impetigo Contagiosa.—The eruption may be pustular from the beginning; the pustules vary in size from a pea to a large marble; they are rounded, firm, and semiglobular; they appear in crops; they are commonly discrete; they show no tendency to break, but dry up and form thin yellow crusts; and they excite but little itching. The disease is contagious and auto-inoculable; occurs especially in children; and lasts from ten days to two weeks.

Varicella, or Chicken-pox.—The pustules result from vesicles; they appear especially on the trunk; they are small and are not umbilicated and they excite but little itching. There is some fever. The disease lasts but three or four days.

Ecthyma.—This disease is observed especially in poorly nourished adults. The pustules vary in size from a pea to a cherry; they are few in number; they are mounted on an inflammatory base, and are surrounded by a distinct inflammatory areola; they excite but little itching; they seldom break, but dry up and form brownish crusts.

Smallpox.—In this disease shot-like papules and umbilicated vesicles precede or are associated with the pustules. The latter are small, surrounded by a red areola, and usually excite some itching. The high fever and history of contagion will assist in making the diagnosis.

Syphilis.—The pustules are frequently associated with other lesions; they are often mounted on a copper-colored inflammatory base; they excite no itching; and they are usually associated with the history and the other evidences of syphilis.

Scabies.—The pustules are small and usually associated with papules, vesicles, and *burrows*; they are especially observed on the hands, forearms, in the axillæ, under the

mammæ, and on the inner aspects of the thighs, and they excite considerable itching. There is often a history of contagion.

Papules.—A papule is a circumscribed solid elevation of the skin varying in size from a pin-head to a pea. Papules are observed in the following conditions:

Erythema Multiforme.—The papules are often associated with macules and tubercles; they are flat, and are of a bright red or purple color; they appear especially on the extremities; and they show no tendency to suppurate, but gradually disappear in the course of two or three weeks; they excite little or no itching, but they are often associated with rheumatoid pains.

After the Use of Certain Drugs.—Bromids, iodids, copaiba, cubebs, and tar may produce a papular eruption. The history will aid in the diagnosis.

Eczema Papulosum.—The papules are very small, closely aggregated, and often associated with vesicles and pustules; the skin is thickened; and there is intense itching.

Miliaria, or Prickly Heat.—The papules are very small; they are very often associated with minute vesicles; they always remain discrete; they appear especially on the trunk; and they excite a little burning and itching.

Acne Vulgaris.—The papules are, as a rule, confined to the face, back, and shoulders; they are usually associated with pustules and comedones; they involve the sebaceous follicles; and they do not excite subjective symptoms.

Scabies.—The papules are small and are usually associated with pustules, vesicles, and burrows; they are especially observed on the hands, forearms, in the axillæ, under the mammæ, and on the inner aspects of the thighs; and they excite considerable itching. There is often a history of contagion.

Syphilis.—The papules are dark in color; they are widely distributed, being especially marked on the trunk and flexor surfaces of the extremities; they are usually associated with

pustules; and they excite no itching. The history and the accompanying evidences of syphilis will aid materially in establishing the diagnosis.

Smallpox.—The papules are hard and have a shot-like feel; they soon terminate in umbilicated vesicles; they excite some itching, and they are associated with high fever, pain in the back, and often a history of contagion.

Measles.—The papules are small, and tend to run together to form crescentic-shaped patches; and they are associated with moderate fever, swollen cervical glands, coryza, conjunctivitis, and bronchitis. There is often a history of contagion.

Tubercles.—Tubercles are large, circumscribed, solid elevations of the skin varying in size from that of a large pea to that of a walnut. They are observed in the following conditions:

Erythema Nodosum.—The tubercles are large; they usually appear on the extremities; they are reddish-purple in color; they never suppurate; and they are associated with malaise, slight fever, and rheumatoid pains.

Erythema Multiforme.—The tubercles are usually associated with macules and papules; they are flat, and are of a bright-red or purple color; they appear especially on the extremities, and they show no tendency to suppurate, but gradually disappear in the course of two or three weeks. They excite no itching, but are often associated with prostration and rheumatoid pains. The disease is probably allied to erythema nodosum.

Lupus Vulgaris.—This may begin as a papule or tubercle. It is especially observed on the face. The tubercles are of a pale-red color and are quite soft to the touch. As a rule, they slowly break down and form shallow ulcers with soft red margins. The ulcers are painless and secrete but little material. They may invade all the soft structures, but the bones escape.

Syphilis.—The tubercular syphilid manifests itself as dark-red tubercles. There are seldom more than three or four,

and they usually appear on the face and extremities. They are very firm and often break down, forming deep, punched-out ulcers that secrete an abundant purulent material.

Tinea Sycosis, or Barber's Itch.—The tubercles appear on the hairy parts of the face and involve the hair-follicles. Suppuration soon begins in the center of the tubercles, and the hairs become dry, brittle, and loose. The microscope will reveal the trichophyton.

Leprosy.—One form of leprosy manifests itself as tubercles. The latter are of a pale-red or yellow color, and undergo slow absorption or ulceration. There is usually more or less anesthesia in the parts affected.

Wheals, or Pomphi.—Wheals are evanescent elevations of the skin, generally more or less round and often white in the center and pale-red at the periphery. They excite considerable itching. They are observed in the following conditions:

Urticaria.—The wheals appear in crops; they are of very short duration; they may appear on any part of the body; and they excite intense itching.

Erythema multiforme, peliosis rheumatica (Schönlein's disease), and certain insects, such as mosquitos, also produce wheals.

Crusts.—Crusts consist of dried exudation, and may be red, yellow, brown, or green in color. They are marked in the following diseases:

Eczema.—The crusts are usually associated with pustules and vesicles; the surrounding skin is red and thickened; and there is considerable itching.

Seborrhea.—Crusts of seborrhea are usually observed on the scalp. Itching is absent, and there are no evidences of inflammation.

Syphilis.—The crusts are thick; they are of a dark-brown or green color; and they are often associated with ulcers that freely discharge. The history and other evidences of syphilis will aid in the diagnosis.

Impetigo.—The crusts are thin and yellow, and they are associated with blebs that appear in crops.

Favus.—The crusts usually appear on the scalp; they are yellow, brittle, and cup-shaped; they are usually perforated by a hair, and have a peculiar musty odor.

Tinea Tonsurans, or Ringworm of the Scalp.—In neglected cases this affection may be associated with crusting. It is observed only in children. The grayish scales, the dry, brittle, and broken hairs projecting through the crusts, the alopecia, and the detection of the trichophyton are the diagnostic features.

Scales.—Scales are dry exfoliations from the upper layers of the skin. They are observed in the following diseases:

Squamous Eczema.—The scales are usually associated with papules; the underlying skin is red and thickened, and there is often marked itching.

Seborrhæa Sicca.—The scales are greasy, and the underlying skin shows no evidence of inflammation. The sebaceous follicles are often dilated.

Psoriasis.—The scales are dry, and are of a pearly-white color; they are associated with circumscribed, sharply defined, elevated, inflammatory patches. The extensor surfaces are especially involved. There is little or no itching.

Ichthyosis.—This affection begins in early life. The scales are dry, and are especially marked on the extensor surfaces. Itching is absent, and there is no evidence of inflammation.

Syphilis.—The scales are dry and are of a grayish color; they are usually associated with papules; and they are especially marked on the palms and soles. There is no itching. The history and other evidences of syphilis will assist in the diagnosis.

Pityriasis Rosea.—The scales are found especially on the trunk, and are associated with small, rose-red macules. There is no itching. The disease runs an acute course of a few weeks' duration.

Ringworm.—The scales are dry and scant; they are associated with circumscribed red patches that tend to disappear

in the center. There is often marked itching. Microscopic examination reveals the trichophyton.

Ulcers.—Ulcers are observed especially in the following diseases:

Syphilis.—The ulcers are deep; they have a punched-out appearance; they secrete an abundant offensive material; they often involve the bone; they extend rapidly; they are not painful, and the imperfect cicatrix which they produce is soft. The history and other evidences of syphilis will aid in the diagnosis.

Epithelioma.—This appears in late life; there is usually a single center of ulceration; the ulcer is irregular in shape; the edges are thickened and infiltrated; the secretion is scanty and bloody; the progress is somewhat slow, and there is often pain.

Lupus Vulgaris.—This usually appears in early life; there are often several centers of ulceration; the ulcers are usually superficial; the edges are not thickened; the progress is extremely slow; the bones are never involved; there is very little secretion, and soft papules often develop in the cicatrix, which is firm and contracted.

Simple ulcers may result from traumatism, the application of caustics, or the action of intense heat or cold. Ulcers are frequently observed on the legs of old persons in association with varicose veins. Simple ulcers may be recognized by the history, location, appearance, and absence of other causes.

Perforating Ulcer of the Foot.—This term is applied to a deep-seated ulcer appearing on the sole of the foot and most frequently observed in locomotor ataxia. It usually begins as a corn in the neighborhood of the great toe, and is generally associated with anesthesia of the sole of the foot.

Decubitus.—This term is applied to the bed-sores that form after the occurrence of grave cerebral or spinal lesions. They are usually observed on parts that are subjected to pressure as the sacrum, buttocks, calves, and heels, and are preceded by erythema and vesication.

NON-INFLAMMATORY DISEASES OF THE SWEAT-GLANDS AND THE SEBACEOUS GLANDS

HYPERIDROSIS

Definition.—Excessive sweating.

Etiology.—As a general condition it is often observed in advanced tuberculosis and in other diseases characterized by marked debility. Local hyperidrosis is most frequently observed in the hands, feet, and axillæ, and probably results from some derangement of the sympathetic nervous system. Unilateral sweating of the face may indicate an aneurysm or tumor pressing on the cervical sympathetic.

Symptoms.—The primary symptom is excessive sweating, and this often leads to intertrigo, prickly heat or eczema. Bromidrosis is often associated with the hyperidrosis.

Prognosis.—The disease is frequently rebellious to treatment.

Treatment.—The underlying condition, if ascertainable, should be treated. Internally, one of the following remedies may be employed to diminish the amount of sweat: belladonna, picrotoxin, or agaricin.

Local Treatment.—Dusting-powders of starch, talc, or lycopodium with tannoform or boric or salicylic acid; or lotions containing formaldehyd (1 to 100), sulphate of zinc, tannin, or alum are often useful.

R.	Pulveris acidi salicylici	
	Pulveris zinci carbonatis præcipitati	
	Pulveris magnesic ustæ.....	āā ʒiv
	Pulveris amyli.....	ʒxv
	Pulveris talci.....	ʒxx.—M.
SIG.	Dusting-powder.	(HARDAWAY.)

In hyperidrosis of the feet the method suggested by Hebra is often very efficient. The feet should be washed, thoroughly dried, and then carefully enveloped in strips of muslin that have been spread with diachylon ointment. The application should be made twice daily. In the dressing no water should be employed, but the feet must be carefully wiped and then dusted with starch or lycopodium before the ointment is reapplied. The treatment should be continued for from one to two weeks, after which the feet may be washed and the dusting-powder alone used.

BROMIDROSIS

(Osmidrosis)

This is a functional disturbance characterized by the secretion of sweat of an offensive odor. It is frequently associated with hyperidrosis, and the feet and axillæ are the parts commonly affected. The treatment is that of hyperidrosis. A 1 per cent. solution of potassium permanganate or a 2 per cent. solution of liquor formaldehydi is often useful. The *x*-ray is sometimes of service.

CHROMIDROSIS

This is a functional affection characterized by the secretion of colored sweat. The parts most frequently affected are the face and trunk; the most common colors are red and yellow. It is often associated with hyperidrosis.

SUDAMEN

Definition.—A cutaneous affection, characterized by the eruption of minute vesicles, resulting from the retention of sweat in the upper layers of the skin.

Etiology.—It is often observed in health in persons who perspire freely. It is frequently noted in febrile diseases that are associated with sweating, such as pneumonia and typhoid fever.

Symptoms.—Minute, irregular, translucent vesicles appear on the surface. They are not surrounded by inflammatory areolæ. They do not rupture, but dry up and are followed by slight desquamation. The affection has little significance, and treatment is rarely required.

SEBORRHEA

Definition.—A functional affection, characterized by excessive secretion of sebaceous material, which forms an oily coating or scales. Two forms are recognized: Seborrhea oleosa and seborrhea sicca.

Etiology.—In many cases the cause is not apparent. Often the disease is associated with impairment of the general health. By some it is regarded as of parasitic origin.

Seborrhea Oleosa.—This form is most commonly observed on the face, particularly about the nose, which is habitually bathed in an oleaginous material that has exuded from the sebaceous follicles. From irritation the parts are often red. The condition is frequently associated with seborrhea sicca, comedo, and acne.

Seborrhea Sicca.—This form is most frequently observed on the scalp, and constitutes what is popularly termed *dandruff*. Examination reveals an incrustation composed of thin, yellowish-gray, greasy scales. In uncomplicated cases the skin is pale, but from irritation it may subsequently become hyperemic or inflamed. When allowed to continue, the nutrition of the hair is interfered with and baldness results.

On the body seborrhea sicca appears as yellowish-gray, slightly elevated patches covered with greasy scales. The outlets of the follicles are often dilated. There is usually more or less redness of the skin from hyperemia (*seborrheal eczema*).

Diagnosis.—In *eczema* the skin is red and thickened; there is marked itching; and the scales are not greasy. In *psoriasis* the scales are dry and pearly and there are evidences of inflammation.

Prognosis.—This is favorable under prolonged and appropriate treatment.

Constitutional treatment is frequently required. Disturbances of digestion, constipation, and faulty nutrition will each require appropriate measures. Systematic exercise, preferably in the open air, the free use of water between meals, and regularity in eating often aid materially in effecting a cure.

In dry seborrhea (*dandruff*) the scales should be removed with warm water and a mild soap. Frequent washing is usually necessary. Crusts may be softened with oil or with oil to which salicylic acid, 5 grains to the ounce, has been added. The most useful local remedies are sulphur, resorcin, ammoniated mercury, and salicylic acid.

R̄.	Ceræ albæ.....	ʒij
	Petrolati liquidi.....	fʒij
	Sodii boratis.....	gr. x
	Sulphuris præcipitati.....	ʒij
	Aquæ rosæ.....	fʒviij.—M.

Ft. Unguentum.

SIG.—Apply at bed time for several nights and then shampoo

R̄.	Resorcinolis.....	ʒij
	Olei ricini.....	℥ x-xx
	Spiritus myrciæ	
	Alcoholis.....	āā fʒ iiij.—M.

SIG.—Apply between the hairs by means of an eye-dropper, using gentle friction.

Ammoniated mercury may be used in an ointment containing from 20 to 30 grains to the ounce. Salicylic acid, 10 to 20 grains to the ounce, may often be added to the mercurial ointment with advantage. In oily seborrhea of the face a lotion of resorcin and boric acid usually acts well.

R̄.	Resorcinolis.....	gr. v-x
	Acidi borici.....	ʒij
	Alcoholis.....	fʒij
	Aquæ rosæ.....	q. s. ad fʒiv.—M.

COMEDO

Definition.—A functional disease of the sebaceous glands, characterized by the retention of discolored sebaceous material in the distended ducts of the glands.

Etiology.—It is most frequently observed in young adults and has the same etiology as acne, with which disease it is usually associated. The mite—*Demodex folliculorum*—often found in the lesions, is of no etiologic significance.

Symptoms.—The disease is characterized by an aggregation of minute black or yellowish spots (*blackheads*) that correspond to the outlets of the sebaceous glands. The lesion is often slightly elevated, and when the skin is squeezed, a white, filiform mass exudes, to which the term “flesh-worm” has been popularly applied. The parts most commonly affected are the face, back, and ears. The condition is frequently associated with acne.

Prognosis and Treatment.—The prognosis is favorable, but relapses are common. The systemic treatment is that of acne (see p. 571).

Local Treatment.—Large plugs may be pressed out by means of a watch-key or a special instrument for the purpose. Softening and removal of smaller plugs may be hastened by the application of cloths wrung out in very hot water. Kneading and the application of alcohol and green soap will also assist in their expulsion. Mercury and sulphur are useful remedies.

℞. Hydrargyri oxidi flavi..... gr. x
 Unguenti aquæ rosæ..... ʒij.—M.

MILIUM

Definition.—An affection characterized by the appearance of small, pearly, non-inflammatory elevations, which result from the accumulation of inspissated sebum in ducts the outlets of which have been occluded.

Symptoms.—It is usually observed about the face, and consists of a collection of small, round, pearly elevations,

which vary in size from a pin-head to a millet-seed. The contents of the distended duct cannot be squeezed out until an opening is made, and thus it differs from comedo. It is frequently associated with comedo and acne.

Treatment.—The lesions should be punctured and the contents squeezed out with a comedo extractor.

STEATOMA

(Wen)

Definition.—A steatoma, or wen, is a cyst resulting from the retention of secretion in a sebaceous gland.

Symptoms.—One or more rounded or oval elevations, varying in size from a pea to a large walnut, slowly appear on the scalp, face, or back. They are painless, somewhat soft, and when opened are found to contain a yellowish-white, caseous mass.

Treatment.—The sac and its contents should be carefully dissected out. Simple excision and evacuation are always followed by a return of the cyst.

HYPEREMIC AND INFLAMMATORY DISEASES OF THE SKIN

ERYTHEMA SIMPLEX

Simple active congestion of the skin may be caused by exposure to heat or cold, to the actinic rays of the sun or to x-rays, by traumatism, by the local action of certain drugs, plants, soaps, etc., or by various visceral or nervous disturbances. It is characterized by diffuse redness, disappearing on pressure, and without thickening or elevation of the skin. When it is marked, there may be slight burning.

The *treatment* consists in removing the cause and applying sedative lotions or dusting powders.

ERYTHEMA INTERTRIGO

(Chafing)

Definition.—Hyperemia induced by the attrition of opposing surfaces of the skin.

Etiology.—It is common in children and in fat subjects, and is especially noted where there are friction and perspiration, as under pendulous mammæ, between the upper parts of the thighs, and around the genitalia.

Symptoms.—It is characterized by diffuse redness, moisture, and a feeling of soreness and burning. If it continues, the epidermis may become macerated and an actual dermatitis may ensue.

Treatment.—After bathing the parts with a lotion of boric acid and drying them, the following dusting-powder may be used:

℞. Pulveris zinci oxidi.	
Pulveris talci veneti.....	āā ʒiij
Pulveris camphoræ.....	gr. v.—M

ERYTHEMA NODOSUM

(Dermatitis Contusiformis)

Definition.—An acute inflammatory disease, characterized by crops of large, bright-red nodes that, in the process of evolution, assume different colors, as in the fading of a bruise.

Etiology.—It is observed most frequently in children and young adults. Digestive disturbances are not rarely associated with it. It is closely allied to erythema multiforme, and an affinity appears to exist between both of these conditions and visceral purpura.

Symptoms.—There is a sudden eruption of bright-red nodes, varying in size from a pea to an egg. The extremities are most commonly affected. The advent is marked by malaise, headache, slight fever, and rheumatoid pains. At first the lesions resemble boils, but, unlike the latter, they do not suppurate, but gradually turn yellow, blue, and green, as a bruise.

Prognosis.—The outlook is favorable, recovery ensuing in from a few days to several weeks.

Treatment.—Saline laxatives and sodium salicylate are recommended. Locally, a lotion of a saturated solution of boric acid or of lead-water and laudanum is useful.

ERYTHEMA MULTIFORME

Definition.—An inflammatory disease characterized by erythematous, papular, vesicular, or bullous lesions.

Etiology.—It is more common in women than in men. It usually develops in the spring or fall. Some form of intestinal intoxication appears to be the causative factor. Erythema

nodosum, urticaria, angioneurotic edema, and visceral (Henoch's) purpura are closely related affections.

Symptoms.—The disease is marked by an eruption, usually on the extremities, of macules, papules, vesicles, or, more rarely, bullæ. The lesions may aggregate or remain discrete; they last one or two weeks and gradually fade. There is usually not much itching. In some cases there is decided constitutional disturbance, manifested by malaise, headache, slight fever, and rheumatoid pains.

Diagnosis.—*Dermatitis Herpetiformis*.—The marked itching, the greater tendency for the lesions to cluster, and the chronic character of dermatitis herpetiformis will usually prevent an error in diagnosis.

Urticaria.—In this disease the individual lesions last a very short time and are associated with marked itching.

Prognosis.—The disease runs an acute course lasting from a few days to several weeks.

Treatment.—Rest and a light diet are indicated. Salicylates are sometimes of service. Constipation should be relieved by saline laxatives. Locally, lotions of boric acid followed by dusting-powders are helpful.

URTICARIA

(Hives; Nettlerash)

Definition.—An inflammatory affection characterized by the eruption of whitish or pale-red, evanescent wheals that are attended by severe itching.

Etiology.—Digestive disturbance is an important predisposing factor. Idiosyncrasy to certain articles of food, such as fish, crabs, lobsters, clams, strawberries, etc., is responsible in some cases. Various drugs, such as quinin, copaiba, coal-tar derivatives, and therapeutic sera may also excite it. An association with certain diseases, particularly asthma, gout, jaundice, primary purpura, and diabetes, is sometimes

observed. The bites of mosquitos, fleas and other insects, and the irritation caused by jelly-fish, caterpillars, etc., may excite urticaria in susceptible subjects. Changes in environment may favor its occurrence. It is not rarely associated with angioneurotic edema.

Pathology.—The disease apparently depends upon a disturbance of the vasomotor nervous system, spasm of the vessels first occurring, and then dilatation with outpouring of serum. In many cases it appears to be an anaphylactic phenomenon.

Symptoms.—The eruption appears suddenly and may be localized or more or less general. The lesions are firm, rounded, pinkish or whitish elevations, surrounded by red areolæ (wheals). They last a few hours and are succeeded by new ones in other places. In some instances wheals of various shapes may be produced by drawing a blunt instrument or even the finger nail over the skin (dermographism).

Varieties.—*Urticaria Papulosa*.—In this form the wheal is followed by a lingering papule that is attended by considerable itching. It is most commonly observed in children.

Urticaria Hæmorrhagica.—The lesions are infiltrated with blood.

Urticaria Tuberosa (Giant Urticaria).—In this form the wheals may reach the size of an egg.

Diagnosis.—*Erythema Multiforme* and *Erythema Nodosum*.—In both of these affections the lesions last much longer, show a predilection for the limbs, and do not often excite much itching.

Prognosis.—In acute cases recovery usually ensues in a few days. In chronic cases the outlook is uncertain unless the underlying cause can be ascertained and removed.

Treatment.—The cause should be sought for and removed if possible. In obstinate cases a diet composed for the most part of milk, farinaceous foods and vegetables, is, as a rule, most suitable. Laxatives, especially salines, are often serviceable. Intestinal antiseptics are sometimes efficacious. In

the acute form a combination like the following may be prescribed:

℞. Acetphenetidini..... gr. xl
 Salophen..... ʒij
 Potassii bitartratis..... ʒiss.—M.
 Fiant chartulæ No. xii.
 Sig.—One every four hours.

Among internal remedies worthy of trial in obscure cases may be mentioned: Calcium lactate (10 grains thrice daily), quinin (5 grains thrice daily), sodium thiosulphate (10 grains three times a day), pilocarpin ($\frac{1}{40}$ grain three or four times a day), and lacto-bacillary cultures. Externally lotions containing phenol and boric acid are very useful:

℞. Phenolis..... fʒj
 Acidi borici..... ʒij
 Glycerini..... fʒss
 Alcoholis..... fʒj
 Aquæ..... q. s. ad fʒviij.—M.

HERPES SIMPLEX

(Fever-Blisters)

Definition.—An acute, non-contagious disease, characterized by groups of small vesicles mounted on inflammatory bases.

Etiology.—Herpes is very common in febrile diseases, especially pneumonia, influenza, malaria, and cerebrospinal meningitis. Local irritation also predisposes to it. It is dependent upon a peripheral toxic neuritis.

Symptoms.—One or more clusters of small vesicles appear, usually on the face or genitalia. The vesicles are mounted on an inflammatory base, contain clear fluid, and show no tendency to rupture. Soon their contents become puriform, dry up, and form reddish-brown crusts that fall off in a few days. Burning and tingling precede and accompany the eruption.

Varieties.—If it appears on the face, it is termed *herpes facialis*; on the genitals, *herpes progenitalis*.

Diagnosis.—The history, the superficial character of the lesion, the burning pain, absence of glandular enlargement and the subsequent course will usually serve to differentiate herpes progenitalis from *venereal sores*.

Treatment.—In facial herpes an ointment of boric acid and menthol will be found efficacious:

R̄.	Acidi borici.....	gr. x
	Mentholis.....	gr. iss
	Petrolati.....	℥ ss.—M.

In herpes about the genitals the parts should be washed frequently, bathed with a saturated solution of boric acid, dried, and then dusted with a powder of calomel (gr. xx) and zinc oxid (℥ j).

HERPES ZOSTER

(Zona; Shingles)

Definition.—An acute inflammatory disease, characterized by groups of small vesicles mounted on inflammatory bases, associated with neuralgic pain, and following the distribution of certain nerve-trunks.

Etiology.—Herpes zoster is probably a specific infectious disease, involving the posterior spinal roots and ganglia. Trauma and exposure to cold and wet predispose to it.

Symptoms.—Clusters of vesicles mounted on inflammatory bases may appear on any part of the body, but they are most frequently observed along the course of the intercostal nerves. Only one side, as a rule, is affected. Sharp neuralgic pain precedes and accompanies the eruption. The fluid in the vesicles soon becomes turbid, dries up, and forms yellow-brown crusts which fall off in a few days.

Prognosis.—The outlook is favorable, neuralgic pains, however, sometimes persist and attacks near the eye occasionally cause permanent damage of that organ.

Treatment.—Analgesics are usually required. Acetphenetidin usually suffices, but recourse to morphin may be necessary.

Local Treatment.—Protective or sedative applications are required. The best are an ointment of menthol (5 grains to $\frac{1}{2}$ ounce of cold cream); a dusting-powder of zinc oxid, powdered camphor, and starch, and a 1 per cent. solution of picric acid.

ACNE VULGARIS

Definition.—An inflammatory disease of the sebaceous glands, characterized by papules and pustules and usually seated on the face or back.

Etiology.—It is most common between the age of fifteen and thirty-five. Anemia, menstrual disorders, and gastrointestinal disturbances predispose. Certain drugs, such as iodid and bromid of potassium and copaiba, may induce the disease.

The *acne bacillus* is apparently the exciting agent, but the *staphylococcus albus* is usually present also in the pustular form.

Pathology.—Acne lesions result from the irritation excited by retained sebaceous matter, hence the papules and pustules are commonly associated with blackheads, or comedones.

Symptoms.—There is an aggregation of small papules, pustules, and comedones about the face, chest, and shoulders. Pustules or papules predominate according as the disease is acute or chronic. New lesions develop as the old disappear, so that the disease usually runs a protracted course. Subjective phenomena are absent.

Varieties.—*Acne Papulosa.*—In this form the lesion reaches the papular stage and advances no further.

Acne Pustulosa.—In this variety the papules develop into pustules.

Acne Indurata.—The inflammation is deeply seated, the base of the papule or pustule is firm, and the lesion is sluggish.

Acne Atrophica.—In this form the lesions are followed by small scars or pits.

Acne Hypertrophica.—In this form there is an overgrowth of connective tissue and the skin becomes thickened.

Diagnosis.—The distribution, the chronic character of the affection, the involvement of the sebaceous glands, and the association with comedones are the diagnostic features which separate acne from all other affections.

Prognosis.—The disease is curable, but is rebellious to treatment and prone to relapse.

Treatment.—Digestive derangements and constipation are important accessory factors in many cases and call for a careful regulation of the diet and appropriate medication. In anemic, debilitated persons such drugs as iron, nux vomica and cod-liver oil (if well borne) are often of service. Outdoor exercise, frequent bathing and free-water drinking are valuable aids to recovery. Vaccine treatment, especially the conjoint use of *Staphylococcus albus* (50 to 500 million) and so-called *Bacillus acne* (3 to 5 million), sometimes gives good results in the pustular form of the disease.

Local Treatment.—In acute cases mild applications, such as the following calamin (zinc carbonate) lotion should be used:

R̄.	Pulveris zinci oxidi.....	℥iij
	Pulveris calaminæ.....	℥iij
	Glycerini.....	f℥ij
	Liquoris calcis.....	f℥vij.—M.

In chronic cases sebaceous plugs should be removed by frequent washing with mild soap and warm water, by thorough sponging with hot water, or, if necessary, by a special comedo extractor. Applications of a stimulating character are required, the best being those containing mercury, sulphur, or resorcin.

Mercury may be used in the form of a lotion or ointment.

R̄.	Hydrargyri chloridi corrosivi.....	gr. ss-ij
	Tincturæ benzoini compositæ.....	f℥j
	Emulsi amygdalæ amaræ.....	f℥iv.—M.
R̄.	Hydrargyri oxidi flavi.....	gr. x-xx
	Unguenti aquæ rosæ.....	℥j.—M.

If there is much pustulation and the lesions are deep-seated an ointment of ammoniated mercury—30 to 40 grains to the ounce—often acts well.

Sulphur may be used in the form of "lotio alba:"

℞. Zinci sulphatis	
Potassii sulphidi.....	āā ʒj
Aquæ rosæ.....	fʒiv.—M.

or Kummerfeld's lotion:

℞. Sulphuris præcipitati.....	ʒiv
Pulveris camphoræ.....	gr. x
Pulveris tragacanthi.....	gr. xx
Liquoris calcis	
Aquæ.....	āā fʒij.—M.

or in ointment containing from $\frac{1}{2}$ to 2 drams of precipitated sulphur to an ounce of lard or cold cream. Resorcin is, as a rule, best prescribed as a lotion containing 5 to 20 grains of the drug to an ounce of water. Boric acid may often be added with advantage.

In refractory cases with deep-seated lesions mild x-ray applications sometimes effect a cure. Repeated applications of the high-frequency current have also been recommended (Stelwagon).

ACNE ROSACEA

Definition.—A chronic affection, usually located on the face in the region of the nose, and characterized by marked hyperemia, dilatation of the vessels, overgrowth of tissue, and acne lesions.

Etiology.—Anemia, menstrual disorders, gastric disturbances, exposure to extremes of temperature, and intemperance are the usual predisposing causes.

Symptoms.—The affected area is of a deep-red color; the vessels are dilated; the skin is thickened and lumpy, and acne lesions coexist. In advanced cases the nose may become extremely large and lobulated (rhinophyma). Subjective phenomena are usually absent.

Diagnosis.—*Lupus Vulgaris.*—In this disease there are soft, pale-red papules, ulceration, cicatrization, and no enlargement of the blood-vessels.

Prognosis.—Unless the hypertrophy is marked, the disease is curable under persistent treatment.

Treatment.—The general treatment is the same as in *acne vulgaris*.

Local Treatment.—Sulphur and mercury are the most reliable remedies. Kummerfeld's solution is useful (see p. 572). Ammoniated mercury ($\frac{1}{2}$ dram to ounce of petrolatum) is also of service. In refractory cases *x*-rays sometimes yield excellent results. Dilated vessels should be destroyed by electrolysis. Large hypertrophies may be removed by the knife.

SYCOSIS

(Simple Sycosis; Folliculitis Barbæ)

Definition.—A non-contagious inflammatory disease of the hair-follicles of the mustache and bearded regions.

Etiology.—The affection results from local irritation and the entrance of pyogenic cocci.

Symptoms.—The disease usually manifests itself on the bearded region of the face, and is characterized by an aggregation of papules and pustules, each of which is pierced by a hair. When the lesions are discrete, the intervening skin is often quite healthy; but when they are close together, it is often infiltrated and hyperemic. During the papular stage the hairs are not loose, but firmly attached; during the pustular stage, however, they can be readily extracted. The pustules show no tendency to rupture, but dry to yellowish-brown crusts. Acute cases are associated with more or less burning and itching. If the disease persists, it may lead to destruction of the hair-follicles, and, as a consequence, to permanent alopecia.

Diagnosis.—*Eczema.*—The lesions in eczema excite severe itching, are not perforated by hairs, and are not confined to the hairy parts.

Tinea Sycosis, or Barber's Itch.—In this affection a red, scaly patch makes its appearance, and is followed by the

development of large, deeply seated nodules. The hairs soon become dry, brittle, and broken off, and can easily be extracted. In doubtful cases the microscope may be employed for the detection of the trichophyton.

Prognosis.—The disease is curable under prolonged and judicious treatment. Relapses are very prone to occur.

Treatment.—In acute cases soothing applications are indicated; thus the parts may be dabbed with black wash or a saturated solution of boric acid, and subsequently spread with oxid of zinc ointment. In chronic cases the crusts should be removed, and the hairs cut close or, preferably, shaved. It is advisable to puncture the pustules and to extract the hairs, so as to preserve the follicles. If the parts are not irritable, stimulating applications are useful, and one of the following may be selected:

R̄. Sulphuris præcipitati gr. xxx-℥iss
 Unguenti aquæ rosæ..... ℥j.—M.

Sig.—Apply twice daily.

R̄. Hydrargyri ammoniati..... gr. xx-xxx
 Petrolati..... ℥j.—M.

R̄. Ichthyol..... ℥j
 Petrolati..... ℥j.

Sig.—Apply twice daily.

Autogenous vaccines are sometimes of service.

FURUNCULUS

(Boil)

Definition.—An acute circumscribed inflammation of a sebaceous gland or hair-follicle, usually terminating in suppuration.

Etiology.—Single boils are usually due to local irritation. The appearance of boils in crops (furunculosis) is frequently indicative of impaired health. The entrance of pus cocci, especially the staphylococcus pyogenes aureus, into the skin is always essential to their production.

Diagnosis.—Furuncles must be distinguished from *carbuncles*; the latter are single, large, flattened at their summits, and have multiple openings.

Treatment.—Any underlying constitutional condition which may be etiologically related to the furunculosis should receive careful attention. Regulation of the diet, the administration of tonics and a temporary change of climate may be valuable aids to recovery in refractory cases. The lesions may sometimes be aborted by an application of mercury and ichthyol:

R. Ichthyolis..... gr. xx
 Extracti belladonnæ..... gr. xxx
 Unguenti hydrargyri..... ℥iss.—M.
 Sig.—Apply locally and make pressure with strips of adhesive plaster.

After the occurrence of suppuration free incision and drainage are indicated.

Secondary inoculations may often be prevented by thoroughly cleansing the contiguous parts with warm water and soap and then bathing them with a solution of corrosive sublimate (1:5000). In recurrent furunculosis the most effective measures appear to be the use of vaccines, preferably autogenous, and the administration of brewer's yeast (a teaspoonful to a tablespoonful three times a day) or of compressed yeast ($\frac{1}{2}$ to 1 cake three times a day before or after meals). Calx sulphurata, in doses of from $\frac{1}{10}$ to $\frac{1}{6}$ of a grain, has also been recommended, but it usually fails.

CARBUNCULUS

Definition.—A circumscribed inflammation of the skin and deeper tissues, characterized by a dark-red, painful node that breaks down and evacuates through several apertures.

Etiology.—The predisposing causes are the same as in furuncle. The condition is especially common in diabetes. The exciting cause is the invasion of several contiguous follicles by staphylococci.

Symptoms.—A dark-red, painful, flattened node appears, surrounded by a dusky-red area of induration. The entire lesion is often several inches in diameter. In a week or ten days suppuration begins, and the contents are discharged through several orifices. There is usually marked constitutional disturbance. The most common seats are the nape of the neck, back, and buttocks.

Prognosis.—Recovery usually ensues, but a fatal result is not uncommon in the aged and debilitated.

Treatment.—Generally, tonics, such as quinin, iron, and whisky, are indicated. Opium may be required to relieve pain.

Local Treatment.—In the early stage they may sometimes be aborted by a central injection of 10 to 20 minims of a 5 or 10 per cent. solution of carbolic acid in glycerin. If not seen until abortion is too late, firm compression may be made by straps applied concentrically, leaving the central orifice free for the discharge of sloughs; an antiseptic dressing may be applied over the straps. As a rule, however, early surgical intervention is to be advised.

PSORIASIS

Definition.—A chronic inflammatory disease, characterized by red, scaly, sharply circumscribed, elevated patches.

Etiology.—Psoriasis usually develops in young adults. Heredity, the gouty diathesis, pregnancy, and lactation seem to predispose. It is as common in the robust as in the debilitated. It is non-contagious.

Pathology.—The lesions consist of a marked hyperplasia of the rete mucosum, thickening of the horny layer, and round-cell infiltration of the corium.

Symptoms.—Little red spots appear on the body and gradually grow until they reach the size of a dime or, perhaps, of a silver dollar. The lesions are of a dull pink or red color, sharply defined, somewhat elevated, surrounded by healthy skin, and covered with abundant dry, pearly, overlapping scales. These scales are readily detached, leaving behind a

dry, slightly excoriated surface. The lesions may be uniformly distributed over the entire body, but usually the extensor surfaces are more affected; a symmetric arrangement is often observed. Itching is slight or entirely absent. After a variable time the center of the patch disappears and leaves behind a spot of healthy skin that gradually increases until no trace of the lesion remains. The disease runs a protracted course of months or years, improving in the summer and growing worse in the winter.

Diagnosis.—*Eczema.*—In this disease the patches are not sharply defined, but shade off gradually into the surrounding skin; there is marked itching; there is usually a decided discharge, and healing begins at the periphery instead of at the center as in psoriasis.

Seborrhea.—In this affection the lesions are usually confined to the scalp and face, while psoriasis is general; the scales are gray and greasy; the patches are not circumscribed and lack the inflammatory character of psoriasis.

Papulosquamous Syphiloderm.—The history, the associated symptoms of syphilis, the coppery color of the lesions, the scant scaling, and the special tendency to involve the hands and soles will render the diagnosis apparent.

Prognosis.—The disease disappears under treatment, but relapse usually follows after a longer or shorter period.

Treatment.—Digestive disturbances, constipation, anemia, etc. should receive appropriate treatment. A low protein diet is believed by some observers (Schamberg, Bulkley) to have a favorable influence on the disease. A mild, warm climate, as a rule, acts favorably. Of special remedies, the most generally useful are arsenic, potassium iodid in increasing doses, alkalis, and salicylates. Arsenic is contraindicated in actively inflammatory phases of the disease.

Local Treatment.—Before active medication is instituted the scales should be removed by warm alkaline baths, followed by friction with soap and water, or an ointment of petrolatum containing 10 grains of salicylic acid (30.0 gm.) to the ounce.

The most reliable local remedies are chrysarobin, tar, resorcin, and ammoniated mercury. Chrysarobin is most suitable for cases in which the number of patches is relatively small. As it stains the skin, as well as the clothing, it should be applied only to the patches themselves, and in no case should it be used about the face, as its entrance into the eye results in severe conjunctivitis. The drug may be prescribed in an ointment containing from 10 to 30 grains to the ounce or in flexible collodion containing about 30 grains to the ounce. Salicylic acid—10 grains to the ounce—sometimes increases the efficiency of the ointment.

Tar is a valuable remedy and may be used in the form of the official ointment diluted, with from 6 to 3 parts of petrolatum or lard, or as oil of cade, similarly diluted. Resorcin is sometimes efficacious. It may be used in ointment containing from 20 to 40 grains to the ounce. Ammoniated mercury is especially useful for lesions about the face and scalp. It is not suitable for large patches on the body. It is applied in ointments of about the same strength as those of resorcin, and a combination with the latter or with salicylic acid not rarely acts well.

Heliotherapy in the form of sunlight or arc light and roentgen radiation sometimes yield excellent results, and may be used advantageously in conjunction with the prolonged baths (several hours) recommended by Hebra.

ECZEMA

(Tetter)

Definition.—An acute or chronic non-contagious inflammatory disease of the skin, characterized by multiform lesions—erythema, papules, vesicles, pustules, scales, and crusts—and associated with infiltration, itching, and more or less discharge.

Etiology.—It is most common in the young and in the aged. Digestive disturbances, debility, and gout predispose to its

development. It may be due to external irritants such as cold, heat, the rhus-plant, hard soaps, certain dyes, etc.

Pathology.—The lesions consist of congestion, with a cellular and serous infiltration of the various layers of the skin.

Varieties.—Eczema erythematosum, papulosum, vesiculosum, pustulosum, squamosum, and rubrum.

Eczema Erythematosum.—This form consists in irregular patches marked by swelling, redness, and slight scaling, and accompanied by itching and burning. The most common seat is the face.

Eczema Papulosum.—In this form there is a close aggregation of minute acuminated papules accompanied by severe itching. It is frequently associated with the vesicular variety. The most common seat is the extremities.

Eczema Vesiculosum.—This consists in an ill-defined red patch surmounted by minute vesicles, and accompanied by intense itching. The vesicles soon rupture and leave a raw, weeping surface that becomes more or less covered with crusts. In children it is most common on the face; in adults, on the extremities.

Eczema Pustulosum (Eczema Impetiginosum).—This consists in an aggregation of small pustules that break and lead to the formation of thick yellowish crusts. Itching is not marked. It is frequently associated with the vesicular variety. It is most commonly observed on the face and scalp of poorly nourished children.

Eczema Squamosum.—In this form there are irregular ill-defined red patches accompanied by considerable scaling. It differs from the erythematous form in the large amount of scaling. Its most common seat is the scalp.

When there is a marked tendency to fissuring, as in *chapping*, this form is termed *eczema fissum*; and when there is a tendency to the formation of warty excrescences, it is termed *eczema verrucosum*.

Eczema Rubrum (Eczema Madidans).—This is a secondary variety and is recognized by a raw, dark-red, moist surface,

more or less covered with thick, yellowish-red crusts. The itching may be severe. In children it is frequently noted on the face, and in old people on the extremities.

Diagnosis.—*Scabies*.—The history of contagion; the location of the lesions—between the fingers, on the wrists, under the mammæ, in the axillæ; and the presence of burrows will indicate scabies.

Psoriasis.—The sharply defined patches, the dry scaling, the absence of marked itching, the symmetric distribution, and the predilection for extensor surfaces will indicate psoriasis.

Acne Rosacea.—The presence of acne papules and pustules and of dilated blood-vessels and the absence of itching will distinguish acne rosacea from erythematous eczema.

Seborrhea.—The greasy scales and the absence of itching and of all inflammatory symptoms will indicate seborrhea.

Sycosis.—The limitation of the lesions to the hair-follicles of the face and the absence of itching will distinguish sycosis from eczema.

Prognosis.—The outlook is favorable under appropriate treatment.

Treatment.—Tonics are often indicated. In strumous children cod-liver oil, if well borne, may be of considerable value. Derangements of the digestive tract are frequently present and will require a regulation of the diet and appropriate medication. Constipation, especially, must be corrected. Gout, diabetes and chronic nephritis sometimes appear to be in etiologic relationship with eczema, and if this is the case suitable treatment must be directed to these conditions. There are no special remedies of much value. Arsenic is occasionally of service in chronic scaly types of the disease, but it is absolutely contraindicated in all acute or subacute phases characterized by bright redness, burning or itching, or oozing.

External Treatment.—In acute cases with pronounced inflammatory symptoms soothing applications should be employed. A saturated solution of boric acid may be dabbed on for several minutes, allowed to dry, and followed by a dusting-powder

of zinc oxid, talc, or magnesium carbonate, or by zinc oxid ointment. The following lotion is also frequently used:

R̄.	Zinci oxidi	
	Calaminæ.....	āā ʒiij
	Glycerini.....	f ʒss
	Liquoris calcis.....	q. s. ad f ʒvii j.—M.

If there is much itching, $\frac{1}{2}$ to 1 dram of phenol may be added to the last lotion.

In all cases of acute eczema soap and water should be used as infrequently as possible.

In subacute eczema salicylic acid, resorcinol, ammoniated mercury or tar may be added to lotions or ointments.

R̄.	Amyli	
	Zinci oxidi.....	āā ʒiij
	Acidi borici.....	ʒij
	Acidi salicylici.....	gr. v-x
	Petrolati.....	q. s. ad ʒj.—M.

In pustular forms of eczema an ointment of ammoniated mercury usually acts well. From 5 to 20 grains may be added to the ounce of zinc ointment or to the ounce of Lassar's paste:

R̄.	Hydrargyri ammoniati.....	gr. v-xx
	Zinci oxidi	
	Amyli.....	āā ʒiv
	Petrolati.....	ʒj.—M.

In chronic eczema crusts and scales should be removed with petrolatum, olive oil, or, if necessary, with starch poultices. The most useful remedial applications are those containing ammoniated mercury, resorcin, salicylic acid and tar.

R̄.	Phenolis.....	gr. x
	Hydrargyri ammoniati.....	gr. xx-xl
	Unguenti zinci oxidi.....	ʒi.—M.

R̄.	Resorcinolis.....	gr. x-xxx
	Acidi salicylici.....	gr. v-x
	Unguenti aquæ rosæ.....	ʒij.—M.

Whatever agent is selected, it is always advisable to use at first a relatively low concentration and to increase the strength cautiously, if the condition requires it, otherwise an irritant rather than a healing effect may be produced.

Finally, in sluggish indurated patches of eczema the roentgen ray sometimes acts very favorably.

LICHEN RUBER AND LICHEN PLANUS

Lichen Ruber.—This is a rare disease, characterized by the eruption of small, red, glazed, acuminate papules that show no tendency to coalesce, and that are associated with itching and failure of general health. The disease runs a chronic course, and may prove fatal through exhaustion.

Lichen Planus.—This form is characterized by an eruption on the extremities of small, red, flat papules that tend to spread, and, by coalescing, form dull-red, irregular patches. The lesions have an angular outline; are slightly umbilicated, and at first have a smooth and shiny appearance, but later are slightly scaly. There is more or less itching, but no impairment of the general health. As the old lesions disappear new ones take their place.

Etiology.—These affections are most frequently observed in poorly nourished, middle-aged males.

Treatment.—The general health must be improved by good food and such tonics as iron, strychnin, and cod-liver oil. Arsenic is of considerable value. Locally, ointments of tar or mercury are useful.

PRURIGO

Definition.—A chronic inflammatory disease, characterized by a general eruption of minute, discrete papules, accompanied by marked itching.

Etiology.—It is most commonly observed in the poor and ill-fed of Europe, and is very rare in America. It develops in early childhood and persists through life.

Symptoms.—An eruption of small, discrete, deeply situated, pale-red papules appears on the body, especially on the back and extensor surfaces of the extremities. The skin is harsh, dry, and thickened, and covered with numerous scratch-marks induced by the intense itching. The disease usually persists through life.

Treatment.—The general health must be improved by good food and the use of nutrient tonics, such as iron and cod-liver oil. Frequent bathing, followed by ointments of tar, sulphur, or naphthol, gives relief.

DERMATITIS HERPETIFORMIS

(Herpes Gestationis; Duhring's Disease)

Definition.—A rare inflammatory disease, characterized by multiform lesions (vesicles, papules, pustules, etc.) forming in groups and accompanied by severe itching, and tending to run a chronic course.

Etiology.—Women are more commonly affected than men. Pregnancy, lactation, and menstrual disorders seem to exert a predisposing influence. The patients, as a rule, are of the neurasthenic type.

Symptoms.—*Erythematous Form.*—This is characterized by the appearance, in crops, of erythematous patches that are associated with considerable itching.

Papular Form.—Groups of papules appear in crops and are frequently associated with erythema, vesicles, and scratch-marks.

Vesicular Form.—Groups of irregularly shaped vesicles resembling herpes appear in crops and are often associated with erythema, pustules, and scratch-marks.

Pustular Form.—This resembles the former, but the vesicles are replaced by pustules.

Bullous Form.—Large, irregularly shaped blebs appear in crops and tend to group. Vesicles and patches of erythema are also frequently present.

Mixed Form.—Vesicles, erythematous patches, pustules, papules, and blebs appear in association, come out in crops, and are attended with intense itching.

In the pustular, bullous, and mixed forms there may be marked constitutional disturbances.

Prognosis.—The disease is resistant to treatment and usually continues indefinitely with remissions and exacerbations.

Treatment.—Arsenic, quinin, and the salicylates are perhaps the most useful remedies. Laxatives are frequently required. Locally, lotions of boric acid or carbolic acid, followed by a dusting-powder of zinc oxid and boric acid, may be employed

to allay itching. Liquor carbonis detergens (a dram to an ounce of camphor water, gradually increased to 6 drams to the ounce) is also useful.

DERMATITIS

Definition.—Inflammation of the skin resulting from the action of some irritant.

Dermatitis Traumatica.—This term is applied to inflammation of the skin resulting from traumatism.

Treatment.—The removal of the cause and the application of soothing remedies will usually suffice.

Dermatitis Venenata.—This term is applied to inflammation of the skin resulting from the application of vegetable, animal, or chemical irritants. Notable examples of this form of dermatitis are observed in susceptible persons after exposure to the influence of poison-ivy (*Rhus toxicodendron*), poison-oak (*Rhus venenata*), or poison-sumach (*Rhus diversiloba*).

Symptoms of Rhus-poisoning.—The affection resembles acute eczema, and may appear in a few hours or not until the lapse of several days after exposure to the plant. It is usually observed on the face or hands. The part becomes red and swollen, and soon minute papules and vesicles appear. The eruption gives rise to considerable burning and itching. As a rule, it subsides in a few days, but in patients with sensitive skin it may linger for several weeks.

Treatment.—The disease is best treated by bathing the affected parts thoroughly but gently with warm water and then applying first a solution of sodium thiosulphate, $\frac{1}{2}$ dram to the ounce, or equal parts of black wash and lime-water, and finally ordinary zinc ointment. Boric acid, 10 grains to the ounce, may be added advantageously to the solution of sodium thiosulphate. A 3 to 5 per cent. solution of potassium permanganate also acts well, but it should not be applied to the face as it stains the skin. Large vesicles should be opened and drained before the medicated lotion is applied.

Dermatitis Calorica.—This term is applied to the inflammation of the skin resulting from extreme heat or cold. *Pernio*, or *chilblain*, is characterized by redness, swelling, intense burning, and itching, and results from a sudden change from a low temperature to a high temperature. *Frost-bite* is characterized by congelation; the part is of a dull-white color and is anesthetic; subsequently inflammation or gangrene develops.

Burns and *scalds* result from the application of dry and moist heat respectively, and are divided into degrees according to the depth to which the destructive process extends.

Treatment.—In *pernio*, or *chilblain*, the part should first be rubbed with snow or bathed in ice-water until the circulation is reëstablished; and then an application made of nitrate of silver (5 grains to the ounce of distilled water) or of tincture of iodine.

In superficial *burns* or *scalds* one of the following remedies may be applied: Carron oil (equal parts of linseed oil and lime-water), powdered bicarbonate of sodium, or:

R. Acidi carbolic. gr. xx
 Petrolati ℥ ij.—M.

Sig.—Spread on lint and apply to the wound.

Dermatitis Medicamentosa.—This term is applied to the various cutaneous eruptions that follow the internal use of certain drugs.

Belladonna or *Atropin*.—These drugs produce a diffuse erythematous rash resembling that of scarlet fever, but it lacks the punctiform character of the latter. It usually appears on the face, neck, and chest, and is associated with dryness of the throat, rapid pulse, and, if the dose has been large, dilated pupils.

Cubeb.—This drug sometimes produces an erythema associated with minute papules.

Copaiba.—The rash may be macular, papular, or like that of urticaria.

Potassium Bromid.—The eruption resembles acne and consists of papules and pustules.

Potassium Iodid.—The eruption may be erythematous, papular, pustular, urticarial, or purpuric. The most common eruption resembles acne, but the lesions are bright red in color and widely distributed over the surface of the body.

Arsenic.—The eruption may be erythematous, papular, vesicular, pustular, or herpetic.

Antipyrin.—This drug not infrequently produces a widespread papular, vesicular, or purpuric eruption.

Quinin.—The rash is usually erythematous, although urticarial, petechial, and bullous eruptions have been observed.

Salicyl Compounds.—The eruption is usually erythematous but it may be urticarial, purpuric or bullous.

Borax.—This drug occasionally produces an eruption resembling psoriasis.

Chloral.—The eruption is usually erythematous or urticarial.

Dermatitis Exfoliativa (Erythema Scarlatinoides; Pityriasis Rubra).—This is a comparatively rare inflammatory disease, characterized by a more or less generalized erythema, followed by scaly or flaky desquamation. Constitutional symptoms are usually mild, but may be severe. The disease may run an acute course of a few days or weeks, or it may become persistently chronic, with exacerbations and remissions. Recurrences are common. The etiology is obscure. It has been observed to follow an attack of eczema or psoriasis, the ingestion of certain drugs and the application of irritant ointments or dressings to the skin. In *eczema* the skin is thickened; oozing is often observed and there is intense itching. The features distinguishing dermatitis exfoliativa from *scarlet fever* are stated on p. 342. The treatment is that of acute or subacute eczema.

ECTHYMA

Definition.—An inflammatory affection, characterized by the appearance of discrete, flat pustules, varying from a quarter to a half inch in diameter.

Etiology.—The disease occurs most commonly in debilitated, poorly nourished adults. It is caused by infection of the skin with pyogenic cocci and is closely related to impetigo contagiosa.

Symptoms.—Flat, yellow pustules appear in crops, especially on the legs. They are surrounded by distinct red areolæ and soon dry up, forming reddish-brown crusts. Slight excoriation and pigmentation sometimes remain after the separation of the crusts. Subjective phenomena are usually absent.

Diagnosis.—The acute course, the absence of ulceration, and the absence of history and of associated symptoms of syphilis will separate it from the *pustular syphilid*.

Impetigo.—This affection is more common in children than in adults and is contagious. The pustules are superficial, without an inflammatory areola, and occur chiefly upon the face and hands.

Prognosis.—The disease is readily cured.

Treatment.—Constitutional treatment is generally required. Such tonics as iron, quinin, strychnin, and cod-liver oil are often indicated.

Local Treatment.—The crusts should be removed and some stimulating ointment applied, as the following:

R̄.	Hydrargyri ammoniati.....	gr. x-xx
	Unguenti zinci oxidi.....	℥j.—M.

PEMPHIGUS

Definition.—A rare, acute or chronic inflammatory disease, characterized by the eruption of successive crops of bullæ or blebs.

Etiology.—General debility from overwork or nervous strain is a predisposing factor. The disease is rare, especially in America.

Varieties.—Pemphigus acutus, pemphigus vulgaris and pemphigus foliaceus.

Pemphigus Acutus.—Fever, chilliness and malaise usually mark the onset. Blebs, varying in size from a pea to an egg, and containing clear or turbid fluid, appear in successive crops and usually develop from the sound skin. In mild cases recovery ensues in the course of a few months.

Pemphigus Vulgaris.—This form usually runs a chronic course, and is characterized by successive crops of blebs, varying in size from a small pea to a large walnut. They are thoroughly distended with fluid, which is at first clear, but subsequently turbid. In some cases the fluid is hemorrhagic. As a rule, the blebs do not rupture, but disappear in the course of five or six days, their contents being gradually absorbed. After absorption a thin pellicle remains, which dries and is subsequently detached, leaving behind a slightly pigmented spot. No part of the body is exempt; and as one set of blebs disappears, new ones rapidly develop, and so the disease continues for many years.

Pemphigus Foliaceus.—This rare and grave form of pemphigus is characterized by crops of blebs, which are flaccid and filled with a turbid fluid almost from the beginning. They soon rupture and form thick crusts, which, separating, leave behind red weeping surfaces. The crops follow each other in rapid succession, and at times the whole body may be covered with blebs and scabs. The disease may last several years, death ultimately resulting from exhaustion.

Diagnosis.—*Bullous Syphiloderm.*—The history, the associated symptoms of syphilis, the thick, yellow, stratified crusts, and the underlying ulceration will serve to separate this affection from pemphigus.

Impetigo Contagiosa.—The acute course, the contagious and auto-inoculable character of the affection, and the umbilication of the blebs will separate impetigo contagiosa from pemphigus.

Prognosis.—The duration and outcome are uncertain. Cases with purulent or hemorrhagic lesions are extremely grave and pemphigus foliaceus is almost always fatal.

Treatment.—The diet should be nutritious, but carefully adapted to the condition of stomach. The patient should be placed under the best hygienic conditions. Tonics, such as iron, quinin, phosphorus, cod-liver oil, and strychnin, are usually indicated. In many cases arsenic is a valuable remedy.

Local Treatment.—The blebs may be punctured and subsequently dressed with an ointment of boric acid.

IMPETIGO CONTAGIOSA

Definition.—An acute, contagious, inflammatory disease, characterized by flat, yellowish blebs that dry up and form thin, yellow, lamellated crusts.

Etiology.—The pyogenic organisms are responsible for the lesions. The disease is most frequently observed in debilitated children.

Symptoms.—The eruption is most frequently observed on the face and extremities; it generally appears in crops, and is at first vesicular. The vesicles grow, and are soon converted into flat, slightly umbilicated pustules which vary in size from a pea to a large walnut. There is rarely any inflammation. Itching is slight or entirely absent. In some cases there is moderate fever with its associated phenomena. In a few days the blebs dry up and form thin, yellow, lamellated crusts that, separating, leave a slightly excoriated surface. The disease is contagious, and the lesions are auto-inoculable.

Diagnosis.—*Eczema.*—In this disease the pustules are deeper, more confluent, excite intense itching, and are associated with inflammation and infiltration of the surrounding skin.

Prognosis.—The disease terminates favorably in from ten days to two or three weeks.

Treatment.—An ointment of ammoniated mercury (10 to 20 grains to the ounce), thoroughly applied, is very efficacious.

To prevent autoinoculation, a lotion of mercuric chlorid (1:5000) may be applied, as well as the ointment, both to the lesions and to the surrounding parts.

MILIARIA

(Prickly Heat)

Definition.—A mild inflammatory disease of the sweat-glands, characterized by the occurrence of minute papules and vesicles.

Etiology.—The affection is due to sweat obstruction and is caused by excessive heat.

Symptoms.—The eruption usually appears on the trunk, and consists of minute, closely aggregated red papules or clear vesicles. The lesions are discrete, and excite some burning and itching. It is usually associated with free perspiration. Recovery occurs within a few days or weeks.

Diagnosis.—*Eczema papulosum* differs from miliaria in that the papules are larger, appear more gradually, disappear more slowly, and excite intense itching.

Eczema vesiculosum differs from miliaria in that the vesicles are large, disappear more slowly, show a tendency to break, and are associated with marked itching.

Sudamen differs from miliaria in that it lacks all inflammatory characteristics.

Treatment.—The general health may require attention. The diet should be light and easily assimilable. Constipation should be relieved by saline laxatives. Locally, a lotion of boric acid, followed by a simple dusting-powder, is usually all that is required.

R.	Pulveris amyli.....	℥vj
	Zinci oxidi.....	℥iss
	Pulveris camphoræ.....	℥ss.—M.
Sig.	—Dusting-powder. (HARDAWAY.)	

ATROPHIC AND HYPERTROPHIC DISEASES

ALBINISM

Definition.—A congenital deficiency of pigment.

Etiology.—Beyond heredity, no cause is known. Partial albinism is more common in the negro.

Symptoms.—In complete albinism the skin is white; the hair is thin, soft and very light in color; the pupils appear red, the eyes are very sensitive to light, and the iris and choroid are deficient in pigment.

VITILIGO

(Leukoderma)

Definition.—An acquired cutaneous affection, characterized by milk-white patches that are surrounded by areas of increased pigmentation.

Etiology.—The disease seems to be more common in the tropics and in the colored race. The condition probably results from disturbed innervation.

Symptoms.—Milk-white spots appear on the body and grow very slowly; their borders usually reveal an increase of the normal pigment. Apart from the absence of pigment the skin is normal. The outlook for cure is unfavorable.

Diagnosis.—*Morphea*.—The initial hyperemia and the subsequent atrophy of the skin will serve to distinguish morphea from vitiligo.

Anesthetic Leprosy.—The subjective symptoms, the atrophy of the tissues, and the anesthesia will separate leprosy from vitiligo.

Treatment.—Tonics and local stimulants may be tried. Among the latter, electricity, blisters, and irritating ointments have been recommended.

ALOPECIA

(Baldness)

Etiology.—(1) Baldness may be congenital; in these cases it is usually partial. (2) It may be an expression of senility, in which case it usually begins on the crown or brow, and is associated with more or less atrophy of the scalp. (3) It very rarely occurs early in life, as an idiopathic affection arising without obvious cause. (4) It often occurs in early adult life as a result of seborrhea. (5) It frequently results from general diseases, such as syphilis, myxedema, typhoid fever and other acute infections.

Prognosis.—In congenital, senile, and idiopathic alopecia the prognosis is unfavorable. In the alopecia of general diseases the prognosis is usually favorable. In alopecia resulting from seborrhea much can be accomplished by persistent and appropriate treatment.

Treatment.—The general health should be improved. Shampooing every one to three weeks with warm water and Castile soap is to be recommended. One of the following local stimulants may be prescribed: Cantharides, quinin, alcohol, capsicum, sulphur, or phenol.

R̄. Tincturæ cantharidis..... f℥ss
 Quininæ bisulphatis..... gr. xv
 Olei ricini..... ℥ss
 Spiritus myrciæ..... q. s. ad f℥iv.—M.

R̄. Tincturæ cantharidis..... f℥iv
 Phenolis..... gr. xx
 Olei ricini..... ℥ss
 Alcoholis..... f℥iv.—M.

If there is much dandruff, the following lotion will be found useful:

R.	Resorcinolis.....	ʒij
	Acidi salicylici.....	gr. xxx-ʒj
	Olei ricini.....	℥x-xx
	Alcoholis.....	q. s. ad fʒvj.—M.
		(SCHAMBERG.)

ALOPECIA AREATA

(Alopecia Circumscripta)

Definition.—Baldness appearing in circumscribed patches without any obvious lesion of the skin.

Etiology.—The cause is unknown. Some regard it as of parasitic origin, while others look upon it as a neurosis. It is usually observed in early adult life.

Symptoms.—The disease is characterized by the sudden or gradual appearance of circumscribed round patches of baldness. At first there is no change in the appearance of the skin, but later it may become pale and atrophied. Although the scalp is the most frequent seat, it occasionally involves other hairy parts, as the eyebrows, beard, etc.

Diagnosis.—*Ringworm.*—Ringworm is rare in adults, and is characterized by elevated scaly patches through which project dry, brittle, broken hairs. If there should be any doubt in the diagnosis, the microscope may be employed to detect the trichophyton.

Prognosis.—In the majority of cases the hair returns under prolonged and persistent treatment. The older the patient, the less favorable the prognosis.

Treatment.—General tonics, such as iron, arsenic, quinin, and strychnin, are usually indicated. The local treatment should be stimulating and consist in the application of blisters, electricity, friction, rubefacient liniments, or ointments containing chrysarobin, tar, sulphur, or ammoniated mercury.

℞.	Tincturæ cantharidis	
	Tincturæ capsici.....	āā f3ss
	Olei ricini.....	f3ss
	Spiritus rosmarini.....	f3ij
	Alcoholis.....	f3j.—M.

Or:

℞.	Betanaphtholis.....	gr. xl-3j
	Petrolati.....	3j.—M.

AINHUM

Ainhum is a rare affection, occurring chiefly in the colored race, and characterized by the appearance of a groove or furrow at the base of one or more of the toes. The groove deepens, the affected member becomes swollen, and finally drops off at the point of strangulation.

LENTIGO

(Freckle)

Definition.—A deposition of pigment in the form of small, irregularly shaped brownish spots.

Etiology.—Blonds are more subject to the affection than brunettes. Exposure to the sun's rays often serves as an exciting cause.

Symptoms.—Exposed parts—the face, shoulders, arms, and hands—are mostly affected. The patches vary in color from yellow to dark brown and range in size from a pin-head to a pea.

Prognosis.—Freckles can be removed, but they always return.

Treatment.—One of the best remedies is the bichlorid of mercury in solution or ointment.

℞.	Hydrarg. chlor. corros.....	gr. ij-iv
	Alcoholis et aquæ.....	āā q. s. ad f3iv.—M.
Sig.—Apply twice daily.		

An ointment containing a dram each of ammoniated mercury and bismuth subnitrate is also useful.

CHLOASMA

Definition.—An abnormal deposition of pigment in the form of large brown or liver-colored patches.

Etiology.—It may result from the application of external irritants; from general diseases like tuberculosis, malaria, exophthalmic goitre, and Addison's disease; or from pregnancy or uterine affections.

Symptoms.—The affection consists in the appearance—especially on the face—of large, round, or irregularly shaped brownish or blackish patches. Apart from the discoloration the skin is normal.

Prognosis and Treatment.—If the cause can be removed, the prognosis is favorable. The best local remedies are bichlorid of mercury, ammoniated mercury and hydrogen dioxid.

℞. Hydrargyri chloridi corrosivi..... gr. ii-j-xij
 Acidi acetici diluti..... f℥ij
 Sodii boratis..... gr. xl
 Aquæ rosæ..... q. s. ad f℥iv.—M.
 Sig.—Apply night and morning.

KERATOSIS PILARIS

(Lichen Pilaris)

Definition.—Small, papular elevations resulting from hypertrophy of the epidermis surrounding the outlets of the hair-follicles.

Etiology.—It usually results from infrequent bathing.

Symptoms.—The skin, particularly on the extensor surfaces of the arms and legs, is the seat of numerous pin-head elevations which have a dirty-gray color and are pierced by hairs. As a rule, there are no evidences of inflammation, but sometimes a few red papules or even pustules result from irritation.

Diagnosis.—In *cutis anserina*, or goose-flesh, the lesions are transient and have the color of normal skin.

Treatment.—In most cases nothing will be required beyond frequent bathing with soap, followed by friction of the skin. In obstinate cases an ointment of salicylic acid (10 grains to the ounce) may be applied after bathing.

MOLLUSCUM EPITHELIALE

(*Molluscum Contagiosum*; *Molluscum Sebaceum*)

Definition.—A cutaneous affection, characterized by the appearance of discrete, wax-like elevations ranging in size from a pin-head to a pea, and varying in color from white to rose-pink.

Etiology.—The disease is usually observed in children, and frequently affects several members of the same household, school, or asylum. It is probably mildly contagious.

Symptoms.—Small white or pale-pink, wax-like elevations appear, especially on the face. They are always discrete and rarely abundant. The center of the elevation is depressed and reveals a dark spot that corresponds to the aperture of the follicle. At first the lesions are quite firm, but as they grow old they become soft. When firmly squeezed, they exude a soft, cheesy material. After remaining for several weeks they break down or undergo slow absorption.

Diagnosis.—The color, the wax-like appearance, the umbilication, and the central aperture are the diagnostic features.

Prognosis and Treatment.—The disease usually yields readily to treatment. General tonics, such as iron, strychnin, and arsenic, are often indicated. The lesions should be incised, the contents expressed, and their bases touched with trichloracetic acid or nitrate of silver. An ointment of ammoniated mercury (40 gr. to the ounce) is sometimes efficacious when the lesions are small.

CALLOSITAS

(Callus; Keratoma; Tylosis)

Definition.—A thickened, horny condition of the skin resulting from hypertrophy of the corneous layer of the epidermis.

Etiology.—Constant irritation from friction or pressure is the chief cause; hence it is frequently seen on the feet from the rubbing of shoes, and on the hands from the friction of tools.

Symptoms.—The condition is characterized by the appearance of hard, thickened, grayish masses, which gradually merge into healthy skin. The soles and palms are the parts most frequently affected. When slight, it causes little inconvenience, but occasionally it becomes fissured and painful.

Prognosis.—It yields rapidly to treatment when the cause is removed.

Treatment.—If excessive, the parts should be soaked and the thickened epidermis pared off. One of the best remedies for softening the horny overgrowth is salicylic acid; it may be applied in the form of an ointment or plaster, or in collodion.

R.	Acidi salicylici.....	ʒj
	Olei ricini.....	℥x
	Collodii.....	fʒj.—M.

SIG.—Apply night and morning.

CLAVUS

(Corn)

Definition.—Clavus is a circumscribed thickening of the epidermis usually appearing on the feet.

Etiology.—Corns usually result from the friction of ill-fitting shoes.

Symptoms.—Small, circumscribed, horny elevations appear upon the feet and often excite severe pain. When bathed in

perspiration, they become more or less macerated, and in this condition constitute the so-called *soft corns*.

Treatment.—A radical cure requires the use of well-fitting shoes. The corns may be removed by soaking, paring, and the application of some mild caustic, such as salicylic acid.

℞. Acidi salicylici..... gr. xxx
Collodii..... f℥ss.—M.

SIG.—Apply night and morning for several days, and then soak in hot water.

℞. Acid salicylici..... ʒj
Emplastri plumbi
Petrolati..... āā℥ss.—M.

SIG.—Apply night and morning.

For soft corns a dusting-powder of salicylic acid (10 grains), boric acid (1 dram) and powdered talcum ($1\frac{1}{2}$ ounce) may be used.

VERRUCA

(Wart)

Definition.—A wart is a circumscribed elevation resulting from hypertrophy of the papillæ and epidermis.

Etiology.—The cause is obscure. A bacterial origin has been suggested. They are most frequently observed in children.

Symptoms.—*Verruca vulgaris*, or common wart, is generally observed on the hands of children. It consists of a firm, circumscribed elevation, varying in size from a millet-seed to a pea.

Verruca plana, or flat wart, is a circumscribed, flat, pigmented elevation usually observed on the backs of old persons.

Verruca Filiformis.—This is a thread-like overgrowth, and is generally observed on the soft parts, like the face and neck.

Verruca Digitata.—This form is made up of numerous branches, and is generally observed on the scalp.

Verruca Acuminata, or *Venereal Wart*.—This appears in groups about the genitalia. It is soft, red in color, and highly

vascular. It may be dry or moist, according to its location; the latter condition often gives rise to a peculiarly offensive odor.

Treatment.—Ordinary warts may be removed by excision, caustics, or electrolysis.

Venereal warts should be bathed in some antiseptic solution and subsequently dusted with calomel, iodoform, or boric acid.

NAEVUS PIGMENTOSUS

(Mole)

Definition.—A circumscribed deposit of pigment, usually associated with hypertrophy of cutaneous structures.

Etiology.—Moles are usually congenital.

Symptoms.—The neck, face, and trunk are favorite localities. The nevi vary in number from one to several hundred; in size, from a millet-seed to a filbert; and in color, from yellow to black. When the surface is smooth, the growth is termed *nævus spilus*; when the surface is covered with hair, it is termed *nævus pilosus*; when the surface is warty, it is termed *nævus verrucosus*; and when there is much overgrowth of connective tissue, it is termed *nævus lipomatodes*.

Treatment.—They may be removed by excision, the application of caustics, or by electrolysis, electrodesiccation or electrocoagulation.

ICHTHYOSIS

(Fish-skin Disease)

Definition.—A chronic affection, characterized by dryness, thickening, and scalliness of the epidermis.

Etiology.—The affection is often hereditary, and is usually detected in early childhood.

Symptoms.—The skin is dry and harsh; the surface is covered with adherent polygonal scales; and the papillæ are more or less hypertrophied. The extensor surfaces of the extremities are the parts most involved.

Diagnosis.—The absence of all inflammatory symptoms will separate ichthyosis from *squamous eczema* and *psoriasis*.

Prognosis.—The disease is incurable, but the patient can be rendered comfortable by appropriate treatment.

Treatment.—The scales may be removed by alkaline baths or by applications of green soap. The skin may be rendered pliable by rubbing in a bland oil or an ointment of salicylic acid (10 grains to the ounce) or of sulphur (20 grains to the ounce). Thyroid extract has been recommended for internal use.

HYPERTRICHOSIS

(Hirsuties)

Hypertrichosis, or hypertrophy of the hair, may be local or general. The term is applied not only to an excessive overgrowth of hair, but to a growth of hair in unusual localities, as on the faces of young women.

Treatment.—The hair may be removed temporarily by shaving, epilation, or depilatories. Permanent relief can be accomplished only by electrolysis or *x*-rays.

SCLERODERMA

(Sclerema; Scleriosis)

Definition.—A pigmented, rigid, indurated condition of the skin, occurring in circumscribed patches or involving a large part of the body.

Etiology.—The cause is unknown. The disease is usually regarded as a trophoneurosis.

Symptoms.—The affection may be diffuse or involve circumscribed patches. It may appear quite suddenly, or develop very gradually in the course of months or years. The skin assumes a yellowish-brown color, becomes rigid, indurated, and hide-bound; the surface is unnaturally dry and smooth. When the condition is advanced, the joints become more or less immobile. An association with Raynaud's disease is somewhat frequently observed.

In the circumscribed form (*morphea*) discrete ivory-like patches, surrounded by a zone of congestion or brownish pigmentation, occur on the trunk or face.

Prognosis.—Recovery may occur, or the disease may be arrested, but not rarely it persists indefinitely.

Treatment.—Internally, arsenic and thyroid extract are the most useful remedies. Locally, warm baths, massage with oily preparations, and electricity in the form of high frequency currents are of service. Injections of thiosinamin have been used with some success. Röntgen-ray treatment is sometimes useful.

ELEPHANTIASIS

(Elephantiasis Arabum; Elephantiasis Pachydermia; Barbadoes Leg)

Definition.—Hypertrophy of the skin and subcutaneous tissues, usually associated with lymphangitis, edema, and pigmentation.

Etiology.—While elephantiasis may occur in any part of the world, it is most common in the tropics. The anatomical cause of the hypertrophic process is inflammatory obstruction of the lymphatic vessels. In tropical cases the obstruction is caused by presence of the *Filaria sanguinis hominis*. Sporadic cases may be the result of the pressure of neoplasms, enlarged lymph nodes, etc., or of repeated attacks of cellulitis.

Pathology.—Examination of the affected tissues reveals hypertrophy of the connective tissue, edema, and inflammation and dilatation of the lymphatic vessels.

Symptoms.—The disease usually begins with recurring attacks of erysipelatoid inflammation. The part is red, swollen, and painful; the lymphatics may be traced as branching red lines beneath the skin; and with these local phenomena there is more or less fever. After each attack the part is left a little enlarged, until finally it presents the following characteristic appearance: it is enormously swollen; the skin is thickened, roughened, and pigmented; and the papillæ are

unusually prominent. The regions generally affected are the legs and genitals. In elephantiasis of the scrotum (*lymph-scrotum*) the hypertrophied mass may weigh as much as 50 or even 100 pounds.

Prognosis.—In the early stage the disease may be arrested, but when fully established, it is incurable.

Treatment.—The acute inflammatory attacks should be treated by rest and the application of sedative lotions, such as lead-water and laudanum. Subsequently mercurial inunctions may be employed, and the part firmly bandaged with the view of promoting absorption. Amputation may be successfully employed in lymph-scrotum. In elephantiasis of the limbs ligation of the main artery has given somewhat encouraging success.

NEW GROWTHS AND INFECTIOUS GRANULOMATA

KELOID

Definition.—A new growth resulting from hypertrophy of the connective tissue of the corium.

Etiology.—It usually results from local injury, though it is claimed that it may arise spontaneously. Certain families and individuals are especially predisposed. It is more frequent in the colored race.

Symptoms.—It begins as a pale-red nodule, which slowly increases in size and sends out claw-like processes. From its resemblance to a crab it has been termed keloid. It is firm, elastic, slightly elevated, sharply defined, and ranges in size from a small bean to a growth as large as the hand. It sometimes excites pain and itching, but usually subjective phenomena are absent. The regions most frequently involved are the chest and back.

Diagnosis.—Keloid may be distinguished from a *hypertrophied scar* by the fact that the latter does not extend beyond the limits of the injury.

Prognosis and Treatment.—The growth is persistent and tends to return after removal. As a rule, the best treatment is excision, followed by x-ray or radium treatments.

FIBROMA

(Fibroma Molluscum, von Recklinghausen's Disease)

Fibroma is a connective-tissue new-growth characterized by the development of variously sized soft or firm tumors in

the skin and underlying tissues. The tumors are painless, range in size from a pea to a hen's egg, and are frequently numerous. The overlying skin may be normal in appearance or slightly hyperemic.

In von Recklinghausen's disease, which is often hereditary, the cutaneous fibromas are numerous and arise from the sheaths of the peripheral nerves. Neuralgic pains, areas of pigmentation, nevus formations, neurasthenic symptoms and feeble-mindedness are not infrequent concomitants.

Treatment.—Growths that cause inconvenience may be removed by excision, the galvanocautery or electrolysis.

ANGIOMA

(Nævus Vasculosus)

Definition.—A new-growth, composed of cavernous tissue or a congeries of small blood-vessels.

Angioma Caverosum.—This form is congenital, is composed of cavernous tissue, and appears as a circumscribed, elevated, dark-red tumor that ranges in size from a pea to one as large as the hand. It is often lobulated and pulsating.

Angioma Simplex (Capillary Nevus; Port-wine Mark).—This form is also congenital, and is composed of a congeries of capillaries. It is non-elevated, bright red or purple red in color, and may cover an area of several inches. It is usually found on the face, and constitutes what is popularly termed a *mother's mark*.

Telangiectasis.—This form is acquired, and is composed of dilated or newly developed capillaries. It appears as a bright red dot from which branch dilated capillaries. It is frequently associated with acne rosacea; it is also common in those of a gouty diathesis and in those much exposed to the weather.

Treatment.—Cavernous angiomas may be removed by ligation, excision, electrolysis or electrodesiccation. Simple angiomas and telangiectasis are best treated by electrolysis or carbon dioxid snow.

XANTHOMA

(Vitiligoidea ; Xanthelasma)

Definition.—A circumscribed connective-tissue new-growth appearing as flat patches or tubercles of a yellowish color.

Etiology.—The condition occurs most commonly in middle life and is more frequent in women than in men. Hepatic disorders, especially obstructive jaundice, seem to exert a predisposing influence. It is sometimes associated with diabetes.

Symptoms.—There are two forms: *xanthoma planum*, which usually appears about the eyelids and consists of smooth, circumscribed, slightly elevated, buff-colored patches; and *xanthoma tuberosum*, which may appear on the neck, shoulders, trunk, or extremities, and consists of small, elastic, and yellowish-colored nodules.

Treatment.—These growths may be removed by excision, electrolysis, or caustics.

EPITHELIOMA

(Skin Cancer)

Etiology.—Late life, heredity, and local irritation are the predisposing factors.

Varieties.—Superficial, deep-seated, and papillomatous.

Superficial Epithelioma (Rodent Ulcer).—This form usually begins as a firm, circumscribed, reddish-yellow, wax-like papule. After the lapse of several months or years the papule becomes scaly, and the removal of the scales is followed by a slight excoriation, which in turn becomes covered with a slight, reddish-brown crust. The latter tends to adhere, and its repeated removal is followed by a raw surface, which is gradually converted into an ulcer. The ulcer has a prominent indurated margin; its outline is irregular; its base is uneven and glazed; and it exudes a sanious, viscid excretion. It is not

painful; it does not lead to enlargement of the neighboring lymphatic glands; nor does it cause impairment of the general health. It spreads very slowly, and sometimes becomes stationary or actually heals. More frequently the ulceration continues until it involves all the tissues of the part, even the bones. The ulcer usually appears on the face, and in its advance it may destroy the nose, eyes, or a large proportion of the cranial bones.

Deep-seated Epithelioma.—This variety may begin as a deep-seated, red, shiny tubercle, or it may develop from the superficial form. The ulcer which is ultimately formed is deep; its base is granular; its edges are everted, indurated, and of a reddish-purple color; it secretes a blood-stained yellow fluid; it is the seat of lancinating pain; it causes enlargement of the neighboring glands; and it sooner or later induces the cancerous cachexia. Death may result from exhaustion, or, more rarely, from hemorrhage caused by ulceration of a large blood-vessel.

Papillomatous Epithelioma.—This may begin as a warty excrescence, or may develop from one of the preceding varieties. It is characterized by an ulcerated surface from which springs an aggregation of large, highly vascular papillæ. Between the papillæ there are often deep-seated fissures from which exudes an offensive viscid discharge. The general health is impaired and the neighboring glands are enlarged.

Diagnosis.—*Lupus Vulgaris.*—Lupus begins in the young; the original papule is soft; there is often more than one center of ulceration; the margins of the ulcer are not hard and everted; the progress is extremely slow; the discharge from the ulcer is very scanty, and the bones are never involved.

Syphilis.—The history, the associated evidences of syphilis, the rapid progress of the ulceration, the abundant discharge, the absence of pain, and the effect of treatment will suggest the diagnosis.

Prognosis.—This should be guarded in rodent ulcer and the deep-seated epitheliomas. A thorough removal in the be-

ginning of the disease is often followed by a permanent cure. When the process is advanced, the growth usually returns.

Treatment.—Epitheliomatous growths may be removed by caustics, the cautery, the curet, electrodesiccation, electrocoagulation, x-rays, radium, or excision.

LUPUS VULGARIS

(*Lupus Exedens*)

Definition.—A local manifestation of tuberculosis, characterized by soft brownish-red tubercles that usually terminate in ulceration and scarring.

Etiology.—The disease usually begins in childhood or early adult life. It is comparatively rare in this country, but common in Europe. The exciting cause is the tubercle bacillus.

Symptoms.—Lupus vulgaris most frequently manifests itself on the face, especially near the nose. It begins as minute, deeply seated, brownish-red papules which grow very slowly until they reach the dimensions of tubercles. These are smooth, quite soft, and seldom painful. The tubercles may either undergo slow absorption or, which is more frequent, break down and leave chronic ulcers. The ulcers are shallow, and their edges are soft and red. There is very little discharge. They spread slowly, and may involve all the soft parts, but the bone is never invaded. While one part of the ulcer is spreading, other parts are being filled with shriveled cicatricial tissue which in turn is often the seat of new tuberculous nodules.

Diagnosis.—*Epithelioma.*—Epithelioma is a disease of advanced life; it begins as a firm, wax-like nodule; the resulting ulcer starts from a single point; its borders are distinctly elevated and hard; it secretes a blood-streaked fluid; and it is often painful.

Syphilis.—The age, history, associated evidences of syphilis, the rapid course, the deep ulcers, the abundant offensive discharge, and later, the involvement of the bones, are the diagnostic features.

Prognosis.—The disease is rebellious to treatment and relapses are common.

Treatment.—The general hygienic measures necessary in the treatment of other manifestations of tuberculosis are applicable to lupus. Tonics are usually indicated.

Local Treatment.—The growth may be removed by cauterization, cureting, excision, electrolysis, or electrodesiccation. One of the following caustic applications may be employed:

R̄. Acidi salicylici..... ℥ss-j
Collodii..... f℥j.—M.

Or:

R̄. Acidi pyrogallici..... ℥ij-iiij
Vasellini et cerati resinæ..... āā'q. s. ad ℥j.—M.
(STELWAGON.)

Often the best results are obtained by cureting and subsequently applying caustics.

Both Finsen light and x-ray therapy have also been used with considerable success in the treatment of lupus.

LUPUS ERYTHEMATOSUS

(Seborrhœa Congestiva)

Definition.—Lupus erythematosus is a chronic disease of the skin characterized by circumscribed red patches more or less lightly covered by yellowish-gray adherent scales and tending to result in atrophic scars.

Etiology and Pathology.—The disease usually begins in early adult life and is much more common in women than in men. Disorders of the sebaceous glands, especially seborrhea, apparently favor its occurrence. The essential cause is unknown, but many observers believe that affection is excited by toxins of tuberculous origin. The characteristic histologic feature is a perivascular infiltration of small round cells in the upper part of corium.

Symptoms.—The disease usually manifests itself on the face, in the region of the nose, and appears as small, red, slightly elevated papules, which are more or less scaly. An erythematous patch is gradually formed by the coalescence of these papules. The periphery of the patch is elevated and sharply defined, while the center is depressed and atrophied. The ducts of the sebaceous glands are dilated and often filled with sebum. The disease spreads very slowly, shows no tendency to ulceration, and rarely excites any subjective symptoms.

Diagnosis.—The location, the sharply defined red patch with an elevated margin and depressed center, the slight scaliness, the dilated sebaceous ducts, the chronic course, and the absence of ulceration are the diagnostic features.

Lupus Vulgaris.—This affection begins earlier in life, is characterized by tubercles and ulceration, and lacks involvement of the sebaceous glands.

Prognosis.—The disease is rebellious to treatment and a complete cure somewhat exceptional.

Treatment.—General tonics, such as iron, arsenic, and cod-liver oil, are often indicated. Quinin seems to be especially useful.

Local Treatment.—In many cases mild applications accomplish the most good. Much benefit is often derived from washing the part thoroughly with green soap and alcohol for a few days and then applying the following lotion:

R̄.	Zinci sulphatis	
	Potassii sulphidi.....	āā ʒij
	Aquæ.....	fʒiij
	Alcoholis.....	fʒj.—M.

SIG.—Shake well, dab the parts for fifteen minutes twice daily, and allow to dry on. (DUHRING.)

In sluggish cases ichthyol (20 per cent. ointment, or 30 to 40 per cent. in water as a lotion) is sometimes useful.

Treatment by the x-ray or carbon dioxid snow has been followed by excellent results in some very obstinate cases.

SYPHILIS CUTANEA

The *secondary symptoms* appear between the first and the fourth month following the chancre, and are characterized by a symmetric arrangement, a coppery color, polymorphism (many forms at the same time), and an absence of itching. They are usually associated with certain general symptoms, such as sore throat, pain in the bones, loss of hair, enlargement of the lymphatic glands, and failure of health.

The *tertiary symptoms* appear in from six months to several years after the primary sore. They are, as a rule, localized, are tubercular, gummatous, or ulcerative in form, and tend to group.

Macular Syphiloderm.—This is a secondary manifestation, and consists in a general eruption of dark-red macules, varying in size from a millet-seed to a ten-cent piece.

Diagnosis.—*Measles.*—The absence of fever, of catarrh, of a crescentic arrangement, together with the history, will prevent an error in diagnosis.

Papular Syphiloderm.—This may be an early or late manifestation, and is characterized by a general eruption of large or small, dull-red papules. A few pustules are also frequently present. It pursues a chronic course, finally disappearing by desquamation, and leaving behind slight pigmentation.

Diagnosis.—The history, distribution, dark color, and the presence of pustules will separate it from keratosis pilaris, papular eczema, and lichen ruber.

Tubercular Syphiloderm.—A late manifestation, characterized by a localized eruption of dark-red, shiny papules varying in size from a pea to a large bean. By some these tubercles are regarded as gummatous in character. They pursue a chronic course and finally disappear by absorption or ulceration. The ulcers thus formed, when single, are round, punched out, and frequently covered with crusts;

when they coalesce, they form a serpiginous sore that pours forth a thick yellowish discharge.

Diagnosis.—*Lupus Vulgaris*.—This occurs in earlier life; it pursues an extremely chronic course; the ulcer is superficial; the tubercles are soft, and frequently redevelop in the scar tissue; the secretion is scant; and the bone is never involved.

Epithelioma.—In this affection the progress is slower; there is only one point of ulceration; the secretion is scanty; and the border is markedly infiltrated.

Bullous Syphiloderm.—This is a late manifestation, and is characterized by an eruption of well-filled blebs varying in size from a coffee-bean to a walnut. The contents of the blebs are puriform. They subsequently form dark, conic, stratified crusts under which are ulcers pouring forth a thick, purulent fluid.

Diagnosis.—*Pemphigus*.—The history, the concomitant symptoms of syphilis, and thick, greenish crusts will serve to distinguish syphilis from pemphigus.

Gummatous Syphiloderm.—This appears as a firm, circumscribed nodule that gradually turns red and softens. It may disappear by absorption, or break down and leave a deep, punched-out ulcer.

Moist Papules (Mucous Patches).—These consist in soft flat papules covered with an offensive, grayish secretion. Heat and moisture favor their development, so that their favorite seats are around the arms, the genitalia, the mouth, and in women under the mammæ.

Papulosquamous Syphiloderm.—This may be an early or late manifestation, and is characterized by a general eruption of small papules that are more or less scaly, so as to resemble psoriasis.

Diagnosis.—The history, the slight scaling, the dirty-gray color of the scales, the dark-red color of the lesions, the especial tendency to involve the palms and soles, will serve to distinguish syphilis from *psoriasis*.

Squamous Eczema.—In this affection the distribution, the infiltration of the skin, and the marked itching will lead to a correct diagnosis.

Annular Syphiloderm.—In this form the lesions consist of circles or semicircles of small, dark-red papules.

Pustular Syphiloderm.—This form usually appears within the first year, and is characterized by a general eruption of small or large, acuminate or flat pustules that finally dry up and form yellowish-brown crusts. Large lesions leave superficial ulcers. The term *rupia* is applied to large, conic, stratified crusts that rest loosely on the ulcerating basis.

Diagnosis.—*Variola*.—Absence of a syphilitic history, of other evidences of syphilis, the shot-like feel, the umbilication, the itching, the high fever, and the acute course will separate variola from syphilis.

Acne.—This is usually limited to the face and shoulders; there is no history of syphilis or concomitant symptoms of that affection.

Treatment.—The internal treatment consists in the administration of antisyphilitic remedies (arsphenamin, mercury, and iodids) and general tonics.

R̄. Hydrargyri iodidi..... gr. j
 Potassii iodidi..... ʒiv
 Syrupi sarsaparillæ compositi
 Aquæ..... āā fʒij.—M.

Sig.—Teaspoonful three times a day after meals.

Or:

R̄. Hydrargyri protiodidi..... gr. v-x
 Extracti opii..... gr. iv.—M
 Fiant pilulæ No. xx.

Sig.—One morning and evening.

Local Treatment.—Papular eruptions may be washed with mercurial lotions; mucous patches may be dusted with calomel; ulcers may be dressed with iodoform.

PARASITIC AFFECTIONS

RINGWORM

Definition.—A contagious disease excited by a vegetable parasite—the small-spore fungus (*Microsporon Audouini*) or the large-spore fungus (*Tricophyton*).

Varieties.—On the scalp it is termed *tinea tonsurans*; on the body, *tinea circinata*; on the bearded region, *tinea sycosis*.

TINEA TONSURANS

This form is observed almost exclusively on the scalp of children. It is characterized by one or more rounded, scaly, elevated, grayish-colored patches through which project dry, brittle, lusterless, broken-off hairs.

Diagnosis.—*Seborrhea*.—The patches are not circumscribed; the scales are greasy; the hair is not involved; and the microscope reveals no parasite.

Eczema.—The patches are not circumscribed; the hair is not involved; there is more inflammation; there is marked itching; and the microscope reveals no parasite.

Alopecia Areata.—Baldness is complete; there are no scales; and the base is smooth and shiny.

Prognosis.—The disease is stubborn, but usually yields to energetic treatment in the course of several months.

Treatment.—Tonics are often indicated. The parts should be thoroughly washed with soap and water, and the affected hairs removed. The following parasitocides may be employed in ointment or lotion: iodine (1 dram to ounce of lard) beta-naphthol, sulphur, chrysarobin, or sulphurous acid.

R̄. Hydrargyri ammoniati..... ʒj
 Petrolati..... ʒj.

SIG.—Apply once or twice daily.

Or:

R̄. Betanaphtholis..... gr. xl
 Sulphuris præcipitati..... ʒj
 Vaselini..... ʒj.—M.

SIG.—Rub into affected area once or twice daily.

(HARDAWAY.)

Treatment by x-rays is commonly very effective.

TINEA CIRCINATA

(Ringworm of the Body)

This appears as one or more rounded, red, slightly elevated scaly patches, which on close examination reveal minute vesicles or papules. As the disease advances new patches spring from the periphery, while the central portion clears up. There is often considerable itching.

Diagnosis.—*Psoriasis*.—The marked scaling; the absence of itching; the tendency to involve the extensor surfaces, especially the knees and elbows; and the absence of the fungus will separate psoriasis from ringworm.

Eczema.—The patches are ill defined; do not clear in the center; there is more infiltration of the skin; and there is no parasitic fungus.

Prognosis.—The disease usually yields rapidly to treatment.

Treatment.—Mercury, sulphur, sulphurous acid, and hyposulphite of sodium are among the best parasitocides.

R̄. Sodii hyposulphitis..... ʒij
 Aquæ..... fʒij.—M.
 SIG.—Apply locally. (DUHRING.)

Or:

R̄. Hydrargyri ammoniati..... gr. xxx
 Adipis..... ʒj.—M.
 SIG.—Apply locally.

TINEA SYCOSIS

(Barber's Itch; Sycosis Parasitica)

This begins as a red, scaly patch involving the bearded region. Soon purplish tubercles and pustules form around the opening of the hair-follicles, and the hairs become lusterless, brittle, and loose. There is often considerable itching.

Diagnosis.—*Simple Sycosis.*—In this the inflammation is superficial; the hairs are not involved; and the trichophyton is absent.

Eczema.—The tubercles, the involvement of the hairs, and the presence of the trichophyton will separate it from eczema.

Prognosis.—Recovery usually ensues in the course of a few weeks.

Treatment.—The affected hairs should be removed, and one of the following parasiticides employed in lotion or ointment: mercury, sulphur, or hyposulphite of sodium.

R.	Hydrargyri ammoniati.....	gr. xl
	Unguenti zinci oxidi.....	℥i
R.	Sodii hyposulphitis	℥iij
	Aquæ.....	℥iij.—M.
Sig.—Apply locally.		
R.	Sulphuris sublimati.....	℥ij
	Petrolati	℥ij.—M.
Sig. Apply locally.		

TINEA VERSICOLOR

(Pityriasis Versicolor)

Definition.—A chronic affection excited by a vegetable parasite, the *Microsporon furfur*, and characterized by fawn-colored scaly patches which usually appear upon the trunk, especially about the chest.

Etiology.—It is a disease of adult life, and is mildly contagious.

Symptoms.—It appears usually on the front of the chest as small round spots of a pale-yellow or fawn color, which

slowly enlarge, fuse, and form slightly elevated, scaly patches.

Subjective symptoms are usually absent.

Diagnosis.—*Chloasma* somewhat resembles *tinea versicolor*, but the former is not often observed on the trunk, is not scaly, and is not associated with a parasite.

Prognosis.—The disease is readily curable, but relapse is not uncommon.

Treatment.—The parts should be frequently washed with soap and water, after which one of the following parasitocides may be applied: Corrosive sublimate (2–3 grains to an ounce of water), sulphurous acid, or hyposulphite of sodium:

℞. Sodii hyposulphitis..... ʒv
Glycerini..... fʒiij
Aquæ..... q. s. ad fʒv.—M.

Sig.—Apply locally.

Or:

℞. Hydrargyri chloridi corrosivi..... ʒj
Alcoholis..... fʒiv
Saponis viridis..... ʒij
Olei lavandulæ..... fʒj.—M.

Sig.—To be rubbed in well night and morning.

(VAN HARLINGEN.)

FAVUS

(*Tinea Favosa*)

Definition.—A contagious affection of the scalp excited by the *Achorion Schönleini*, and characterized by yellowish, cup-shaped crusts.

Etiology.—It is observed especially in poor, ill-nourished children. It is rare in America.

Symptoms.—The disease is characterized by one or more rounded, yellow, cup-shaped crusts, through which project dry, brittle, lusterless hairs. The underlying tissue is more or less atrophied and scarred. It is associated with some itching and a peculiar musty odor.

Diagnosis.—The yellow, cup-shaped crusts, the odor, and the atrophy of the skin will separate it from *ringworm*.

Prognosis.—Favus is very resistant to treatment and is not rarely followed by permanent areas of baldness.

Treatment.—The crusts should be removed by oil or soap and water. The affected hairs should also be removed. The following parasitocides are efficient: mercury, sulphur, chrysarobin, and hyposulphite of sodium. X-ray treatment often gives good results.

SCABIES

(Itch)

Definition.—Scabies is a contagious disease excited by an animal parasite—the *Acarus scabiei*—and manifested by papules, vesicles, pustules, burrows, and intense itching.

Etiology.—The disease is always acquired through intimate intercourse with patients already affected.

Symptoms.—The disease manifests itself by intense itching, which is associated with an eruption of small papules, vesicles, and pustules. Among these lesions may be found cuniculi, or burrows (zig-zag tracks); these are discolored, dotted, slightly elevated lines, ranging from a line to half an inch in length, and produced by the penetration of the female acarus and the deposition of her eggs along the passage. The parts most commonly affected are the hands, between the fingers, the wrists, the axillæ, the genitalia, beneath the mammæ, and the inner aspects of the thighs. The face and scalp are never involved.

Diagnosis.—The recognition of scabies rests on the history, the itching, the presence of burrows, the multiformity of the lesions, and their peculiar distribution.

Prognosis.—The disease is, as a rule, readily cured.

Treatment.—Ointments of sulphur, styrax, and naphthol are efficient remedies. After a thorough bath the whole body should be anointed twice daily for three or four days. At the end of this time the bath should be repeated, and the bed-linen

and underclothing changed. The infected clothing should be sterilized.

R̄.	Sulphuris sublimati.....	℥j
	Balsami Peruviani.....	℥ss
	Adipis.....	℥j.—M.
Sig.—	Rub in thoroughly twice daily.	(DUHRING.)
R̄.	Balsami stryacis.....	℥iv
	Adipis.....	℥iss.—M.

PEDICULOSIS

(Phtheiriasis)

Pediculosis Capitis.—This form results from the pediculus capitis, or head-louse, a gray insect from one to two millimeters in length. The condition is recognized by itching of the scalp and the discovery of the lice or their white ova, or nits. Eczematous lesions resulting from scratching are often observed.

Pediculosis Corporis.—This form results from the pediculus corporis, pediculus vestimenti; or body-louse, a somewhat larger insect than the head-louse. The condition is recognized by intense itching on the covered parts of the body, scratch-marks, petechiæ caused by the bite of the insect, and the discovery of the lice on the garments.

Pediculosis Pubis.—This form results from the pediculus pubis, or crab-louse, a minute, gray, translucent insect. It is found on parts covered with short hair, as the pubes, axillæ, eyebrows, etc.

Treatment.—In *pediculosis capitis* the head may be thoroughly treated with coal-oil, dilute carbolic acid (1 dram to 1 pint), or ointment of ammoniated mercury (20 grains to the ounce), or tincture of cocculus indicus.

In *pediculosis corporis* the parts should be thoroughly washed and the clothes subjected to a high temperature. The body may be bathed in a weak solution of corrosive sublimate.

In *pediculosis pubis* a lotion of corrosive sublimate (2 grains to 1 ounce) or an ointment of ammoniated mercury (1 dram to 1 ounce) is very efficient.

NEUROSES

The most important cutaneous neurosis is pruritus, but neuralgia, hyperesthesia, and anesthesia are also frequently observed as symptoms of various functional and organic nervous diseases.

PRURITUS

Definition.—Pruritus is a functional affection, characterized by itching which is unassociated with any objective phenomena.

Etiology.—Pruritus may arise without obvious cause, as the *pruritus senilis* observed in the old, and the *pruritus hiemalis* which develops on the approach of cold weather and disappears when the weather becomes warm.

Symptomatic Pruritus.—Pruritus may be a symptom of many conditions, notably diabetes, gout, hysteria, neurasthenia, and chronic nephritis.

Symptoms.—There is only one symptom, and that is itching; but as a result of scratching, the part may become hyperemic, thickened, or the seat of eczema.

Diagnosis.—Pruritus must be distinguished from the itching induced by *pediculosis* or some local disease, such as *eczema*.

Prognosis.—This will depend upon the cause. If the primary disease is curable, the prognosis for permanent relief is favorable. In other cases temporary relief only is to be expected.

Treatment.—Search should be made for the exciting cause, which should be removed, if possible. In all cases the urine must be examined for sugar, since diabetes is one of the most

frequent causes of pruritus. Among the internal remedies recommended for pruritus may be mentioned nux vomica, belladonna, and pilocarpin. The best local remedies are phenol, vinegar, thymol, chloral-camphor, boric acid, resorcin, menthol, hydrocyanic acid, and hot water.

R̄. Resorcinolis..... gr. xv-xxx
 Sodii chloridi..... gr. xv
 Glycerini..... f℥ij
 Liquoris calcis..... q. s. ad f℥iv.—M.
 (HARTZELL.)

R̄. Phenolis..... ℥j-iiij
 Glycerini..... f℥ij
 Alcoholis... f℥ij
 Aquæ..... q. s. ad Oj.—M.

R̄. Phenolis..... gr. xv
 Hydrargyri chloridi mitis..... gr. xx
 Unguenti zinci oxidi..... ℥j.

SIG.—Apply locally in pruritus ani.

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